The Journal of HEREDITY

SEGREGATION FOR THE YELLOW BURLEY CHARACTER IN TOBACCO
(SEE PAGE 222)

Mahogany Mice

Talpid^2—Lethal in Fowl Embryos

Naming the Human Chromosomes

An Index of Consanguinity Based on Surnames

Progressive Multiple Lipomatosis in Aging Dairy Cattle

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CONTENTS

STUDIES WITH TALPID^2, AN EMBRYONIC LETHAL OF THE FOWL
By Ursula K. Abbott, Lewis W. Taylor and Hans Abplanalp 195

CHROMOSOME NUMBERS OF POINSETTIA
By L. C. Ewart and D. E. Walker 203

THE INHERITANCE OF MOTTLED LEAF IN COTTON
By Charles F. Lewis 209

A PROPOSED STANDARD SYSTEM OF NOMENCLATURE OF HUMAN MITOTIC CHROMOSOMES
By The Human Chromosome Study Group 214

DIPLOIDIZATION IN *Nicotiana tabacum*
By Bill J. Stines and T. J. Mann 222

MAHOGANY, A RECESSIVE COLOR MUTATION IN LINKAGE GROUP V OF THE MOUSE
By Priscilla W. Lane and Margaret C. Green 228

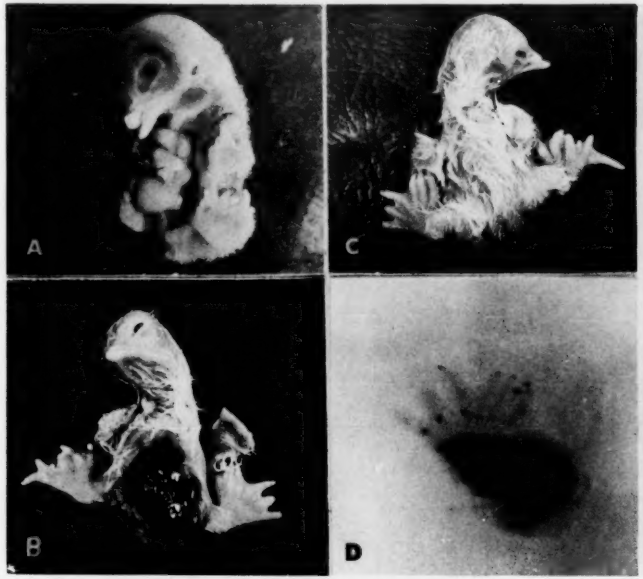
MULTIPLE LIPOMATOSIS IN DAIRY CATTLE
By J. L. Albright 231

Notes and Reviews

AN ALTERNATIVE EXPLANATION FOR SEX REVERSAL AND ABNORMAL SEX RATIOS IN THE FOWL
By Earl H. Newcomer and Grace M. Donnelly 208

NOTES ON A STAMINATE GRAPE SELECTION AS A PARENT
By N. H. Loomis 212

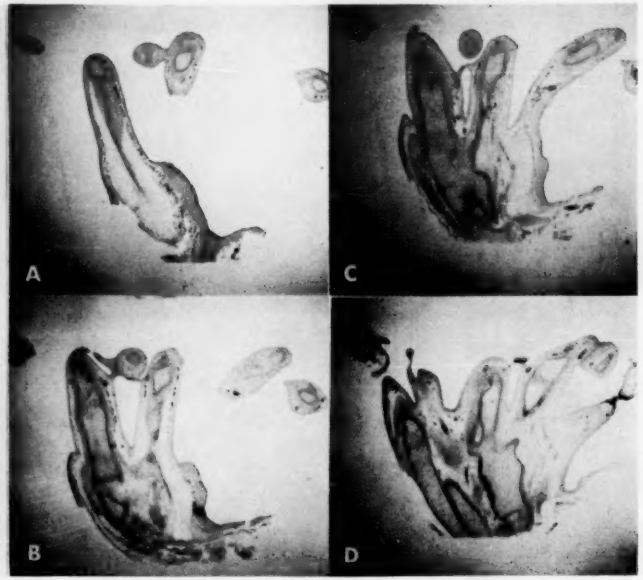
AN INDEX OF CONSANGUINITY BASED ON THE USE OF THE SURNAME IN SPANISH-SPEAKING COUNTRIES
By Richard F. Shaw 221



CHARACTERISTIC TALPID² EMBRYO

Figure 1

A—This embryo, which died on the fourteenth day of incubation, has five digits plus three rudimentary ones on the right foot and seven digits plus two rudimentary ones on the left. Both wings have more than six fused digits. The embryo also manifests dwarfed, shortened dystrophic legs, reduced premaxilla, retarded feathering, subcutaneous edema and hemorrhage, and celosomia (1.5×). *B*—Ventral view (.9×). *C*—Dorsal view (.9×). *D*—An alizarin preparation of the left posterior limb of a talpid² embryo which died on the seventeeth day of incubation shows the change in the pattern of both limbs, as well as the extreme retardation in osteogenesis (1.5×).



ABNORMALITIES IN THE FOOTPLATE

Figure 2

These photomicrographs of the foot of a 10-day talpid² embryo illustrate abnormal relationships of digits and associated tarso-metatarsal bones, absence of any normal digits, syndactylous formation, presence of incomplete digits (often consisting of only a hollow pouch of skin), and delayed ossification (12×).

STUDIES WITH TALPID², AN EMBRYONIC LETHAL OF THE FOWL

URSULA K. ABBOTT, LEWIS W. TAYLOR AND HANS ABPLANALP*

A LETHAL abnormality of the feet and wings was found in 1953 among unhatched chicken embryos from two different but related lines of breeding, described by Lerner[7]. The first line was selected for long shanks (Size Line), but in the second, a derived line, selection for shank length was suspended (Relaxed Size Line). The abnormal embryos had seven or eight syndactylous digits per foot and four or five fingers per wing. In other characteristics as well they resemble Cole's talpid lethal[3].

In a succession of four weekly hatches, four hens mated individually to two different cocks produced abnormal embryos of the above type. The embryos varied primarily in the age at which they died, in the degree of multiplicity of toes and fingers, and in the frequency of their occurrence among normal embryos. One of the above matings was characterized by extremely low hatchability of fertile eggs, suggesting that some embryos died before the abnormality could be detected. Of 14 abnormal embryos found at this time, five were males, three were females and six died before their sex could be definitely established, indicating that the lethal condition was not sex-linked.

Of the four matings producing abnormal embryos, one was of the Size Line (♂ J 66 × ♀ J 1371) and three were of the Relaxed Size Line (♂ J 42 × ♀♀ J 1356, J 1460 and J 1469). Examination of pedigree records of these birds revealed three common ancestors (♂♂ F 76 and F 78, ♀ F 570) in the 1949 generation of the Size Line. An inspection of dead embryo descriptions, obtained routinely for all eggs that do not hatch, indicated that this abnormal syndrome had not been found previously in the Size Line or its derivative, nor had it appeared in the Production Line from which the Size Line was extracted in 1938. The first detectable cases therefore appeared in 1953.

The six known "carriers" were remated with the two males exchanged, the Size Line male being mated to the Relaxed Size Line females and vice versa. In addition, ♂ J 66 of the Size Line was mated to his dam, ♀ H 521. All of these matings produced at least one embryo with the lethal phenotype.

These preliminary results, together with the pedigree analysis, suggested that a single autosomal recessive gene was responsible for the condition and that this gene arose as a mutation in the Size Line at some time between 1938 and 1949. Since breeding stock known to carry Cole's talpid mutation is no longer available†, it has not been possible to test the genetic identity of the new mutation and talpid. Accordingly, this phenotype will be designated as talpid² and the gene as *ta*².

In 1954 the following studies were undertaken with talpid²:

a) tests of the postulated mode of autosomal recessive inheritance;
b) test of linkage of *ta*² with the gene for diplopodia, *dp*[11];
c) test of the integrity of the *ta*² gene and its expression in a genetic background different from that of the Size Line (from the crosses into *dp*-carrier stock);

*Department of Poultry Husbandry, University of California, Davis and Berkeley. The authors are indebted to Mr. R. M. Craig for his able assistance in preparing illustrative material.

†Cole, R. K. Personal communication.

d) description of anatomical and developmental characteristics of the syndrome; features talpid[2] and talpid have in common.

Results

A. *Segregation Data*

Material from two sources was analyzed genetically following the initial test matings reported above. The study of talpid[2] segregations in the Size Line and the Relaxed Line was continued. In addition, the *ta*[2]-carrier females identified in 1953 were outcrossed to two males from an unrelated line carrying the recessive lethal gene for diplopodia, *dp*. This gene, when homozygous, increases the number of foot and wing digits, reduces the length of the premaxilla, decreases body size, and retards rate of development[11, 1].

The matings of the $+dp$ males with the $+ta^2$ females gave rise to only normal phenotypes among 216 identified embryos.

Eleven female and three male progeny of this cross were progeny tested in 1954. One male, mated with his 11 sisters, produced 287 normal progeny and 46 embryos which died before phenotypic classification for talpid[2] or diplopodia was possible (in this case death occurred before 48 hours of incubation). Three females were responsible for the majority of this early mortality. Subsequent tests of these females with other males as indicated below suggest that a lethal unrelated to *ta*[2] was responsible. When a second F_1 male from the outcross was mated with these females, both talpid[2] and diplopod embryos were produced (Table I, 1954). The third F_1 male was tested with $+dp$ females and with $+ta^2$ females from the Size Lines. He produced only normal progeny in the first instance and both normal and talpid[2] offspring in the latter (Table I, 1954).

On the basis of the above F_2 and backcross progenies, 12 F_1 birds were identified genetically. The observed distribution of genotypes supports the presumption that *ta*[2] is an autosomal recessive (Table II).

If the *ta*[2] and *dp* loci are independent, the expectation of nonlethal genotypes is 1 noncarrier : 2 *dp* carriers : 2 *ta*[2] carriers : 4 double carriers. The observed ratio of genotypes among tested normal progeny of double carrier matings agreed closely with this hypothesis (Table II), $\chi^2 = 2.55$ ($p \sim 0.5$). If *ta*[2] and *dp* are closely linked, matings among double carriers should produce 50 percent normal progeny and 25 percent each of the two lethal phenotypes. All normal survivors should be double carriers. Although crossovers could give rise to single carriers, it can be concluded that the presence of noncarriers and a substantial number of single carriers in the progeny of double carrier matings is strong evidence for independence of the two genes (Table II).

Assuming then that *ta*[2] and *dp* are independent autosomal recessive genes, the expectation of phenotypes from matings of double carriers is 9 N : 3 T : 3 D : 1 DT, if the double homozygote is distinguishable from either single homozygote, and 9:3:4, if it is not. Thus, approximately 19 percent of each of the lethal phenotypes and six percent of the double homozygote class are expected in the first instance and 19 percent of one lethal phenotype and 25 percent of the other in the second case.

TABLE I. Incidence of diplopod and talpid[2] embryos in matings segregating for one or both genes

Year	Type of mating: $+dp+ta^2 \times +dp+ta^2$				$+dp+ta^2 \times +++ta^2$			$+++ta^2 \times +++ta^2$			$+dp+ta^2 \times +dp++$			$+dp++ \times +dp++$		
	No.	%U*	%D†	%T‡	No.	%U	%T	No.	%U	%T	No.	%U	%D	No.	%U	%D
1954	59	16.9	4.1	18.4	68	5.9	15.6	....	....	....	348	3.4	20.2	156	1.9	21.6
1955	102	2.0	20.0	27.0	74	2.7	26.4	....	....	....	919	3.2	21.8	....	....	....
1956	413	3.9	16.4	25.7	314	5.4	24.2	....	....	....	532	3.8	23.6	409	3.4	22.3
1957	576	5.2	19.2	24.0	85	16.5	29.6	30	6.7	21.4	406	6.9	27.8	28	7.1	30.3

*% unidentified early deads (of total embryos).
†% diplopod embryos (of total classified phenotypically).
‡% talpid[2] embryos (of total classified phenotypically).

TABLE II. Genotypes identified by progeny test

Genotype	F_1 genotypes in progeny of $++ \; +ta^2 \times +dp \; ++$ matings		Nonlethal genotypes in progeny of $+dp \; +ta^2 \times +dp \; +ta^2$ matings	
	No. observed	No. expected	No. observed	No. expected
$+dp \; ++$	2	3	1	3.4
$+dp \; + \; ta^2$	3	3	13	13.8
$++ \; + \; ta^2$	2	3	9	6.9
$+dp \; ++$	4	3	7	6.9
$+dp$?? *	1	—	1	—

*No test with mates of $+ta^2$ genotype.

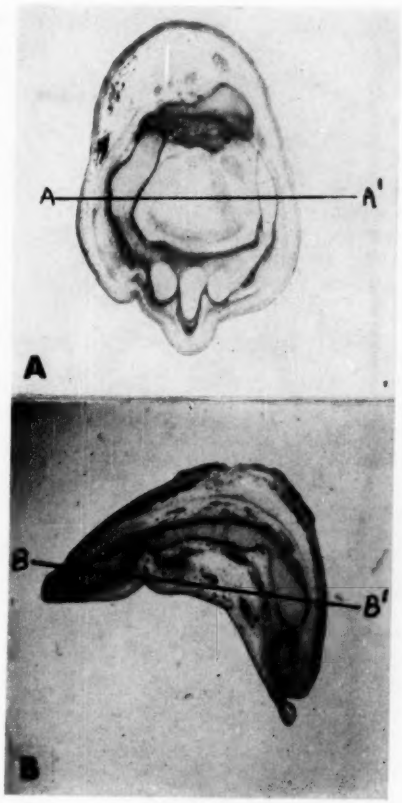
The consistently higher proportion of talpid^2 over diplopod embryos (Tables I and III) in progeny of double carrier matings suggested that the two genes are independent and further that the double recessive homozygote exhibits the talpid^2 rather than the diplopod phenotype. Data of 1954 (Table I) do not preclude the possibility that an unrecognizable double recessive class is included in the high proportion of unidentified early-dead embryos*. Data obtained in the three subsequent years, however, indicated that the proportion of unidentified early-dead embryos obtained was never large enough to account for the numbers expected in a *dpdp ta^{2}ta^2* class.

If *dp* and *ta^2* segregate independently and the *dpdp ta^{2}ta^2* class are talpid^2 in phenotype, the expectation of phenotypes is 9 N : 3 D : 4 T. The χ^2 value for deviations from this expectation (based on Table III, line 1) is 1.35 ($p \sim 0.5$) and is not significant. If the hypothesis of complete linkage is tested with the same data, the deviation of the observed proportion of normal progeny from expectation is highly significant; $\chi^2 = 13.24$ ($p < 0.001$). Table III (lines 2 and 4) also indicates that talpid^2 phenotypes occur in the expected frequency range when only one class of abnormal embryos is expected. The same close fit of observed homozygous recessives to expected frequencies was found for diplopod embryos (Table III, lines 3 and 5).

Sex linkage of the *ta^2* gene is excluded on the basis of the analysis of segregation data by sexes (Table IV) and by the continuing occurrence of phenotypically normal *ta^2*-carrier females.

B. *Morphology and Development*

The entire footplate is altered in talpid^2 embryos (Figures 1 and 2). Each foot has seven or eight syndactylous digits of approximately equal length. The five or six digits on each spatulate wing are fused to form a hand-like structure (Figures 1 and 3). The long bones of the legs and wings are abnormally short and dystrophic. The shortened spinal column is relatively straight rather than curved (Figure 4). The majority of talpid^2 embryos have an incomplete ventral body wall with associated malposition and protrusion of viscera (celosomia). This condition is illustrated in Figure 1 and also in Figure 4. The premaxilla is frequently reduced and occasionally completely absent. In the former instance the egg tooth may occupy an abnormal ventral position. Many talpid^2 embryos have crossed beaks. Variable degrees of subcutaneous hemorrhage and edema are present. Talpid^2 embryos are delayed as much as two days in their rate of development. The retardation is most pronounced in early limb differentiation and growth. Feathering is retarded and abnormal. Total body size is reduced; talpid^2 embryos are generally less than one-half the size of normal siblings of comparable age. Many die between the tenth and fourteenth days of incubation [the average age at death is 13 days (Table IV)] but some survive until the seventeenth or eighteenth day of incubation. With the exception of the 1954 matings, early mortality in talpid^2 embryos was not excessive and did not result in ratios deficient in proportion of talpid^2 progeny, as was the case with Cole's talpid.

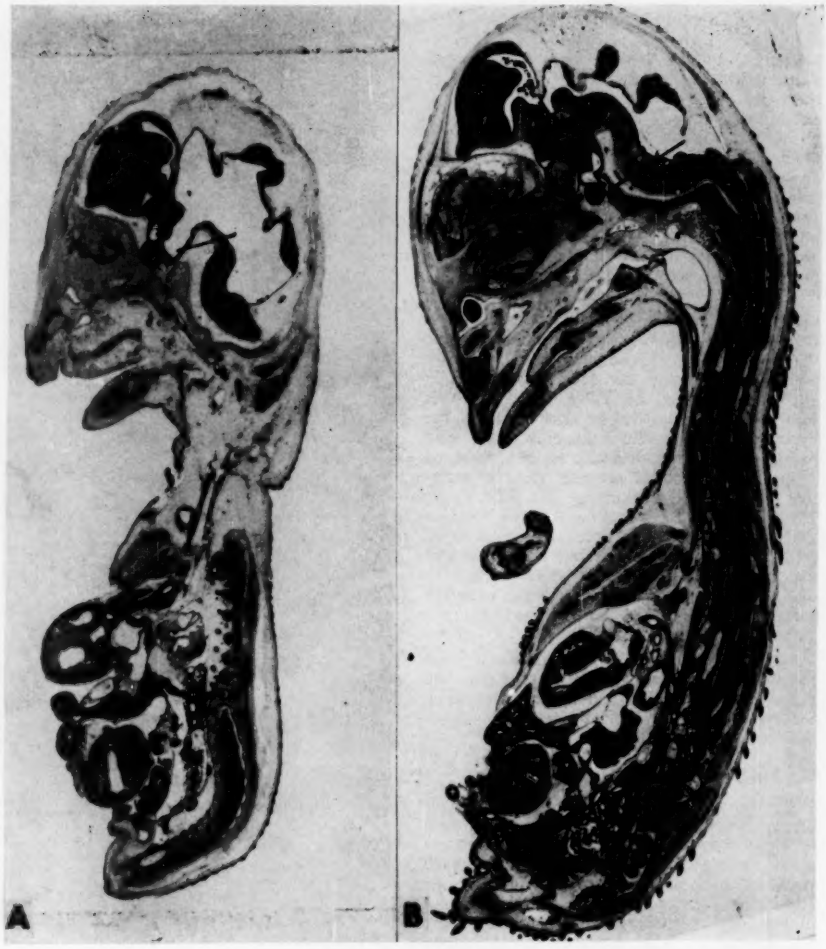


ANOMALOUS WING

Figure 3

Transverse (*A*) and frontal (*B*) views of the wing of a talpid^2 embryo of 10 days' incubation. Relationship of cuts is indicated by A-A' and B-B'. Note the structure of the wing with fused bone and cartilaginous areas and delay in ossification. *A*—28.8×. *B*—25.3×.

*The relatively high incidence of early embryo mortality in 1954 is due largely to inbreeding.



SAGITTAL SECTIONS

Figure 4

Talpid[2] (*A*) and normal (*B*) embryos of 10 days' incubation (6×). The talpid[2] embryo is retarded and reduced in size, especially in the trunk region. Abnormal curvature and the failure of the ventral body wall to close produces displacement and protrusion of viscera (celosomia). The neck is shortened and thickened; the beak and facial parts are highly abnormal; the maxilla is vaulted and shortened, while the tongue is reduced. Calcification of bony parts is retarded as is the formation of feather follicles. Subcutaneous edema is noticeable in dorsal areas of the neck and trunk.

While talpid[2] embryos vary in numbers of digits, body size and involvement of other organs, intermediate or normal-like expressions have not been observed, in contrast to the wide range of expressions in diplopodia[2].

Among phenotypes classified as talpid[2], a small proportion had a footplate with the typical talpid[2] appearance, but which appeared to be separated into two parts; in this respect these embryos suggested the arrangement in diplopodia, in which normal digits II, III and IV form one group and supernumerary digits

HYPOPHYSIS

Figure 5

Magnified sections of the hypophysis in Figure 4*A* and *B*. *A*—100×. *B*—73×.

develop separately in a pre-axial location. It is possible that embryos of this type are *dpdp* ta^2ta^2 (Figure 6).

C. *Histology*

Inman[6] studied the histology of 13 talpid embryos. Among other anomalies, she reported that talpids lack a normal hypophysis, that Rathke's pouch is absent or involved in the formation of supernumerary ectopic lenses, and that the roof of the diencephalon contains numerous epiphyseal structures; also that the limb buds, while showing overgrowth as early as four days, are markedly retarded in differentiation and that supernumerary digits fail to appear by the end of the eighth day of incubation.

In the present study the histology of the brain and associated structures and of the limbs was investigated at 2-, 4-, 5-, 8-, 9-, and 10-day stages. In 2-day stages it was not possible to recognize embryos definitely as talpid^2 for lack of diagnostic features. However, because of the effect on developmental rate found in all later stages of talpid^2, retarded 2-day embryos were designated as "possible talpid^2." This class is likely to contain some non-talpid^2 embryos retarded in growth rate for causes unrelated to the phenotype under study. Possibly Inman's 2½-day stages were subject to the same source of error.

Among 16 talpid^2 and two "possible talpid^2" embryos examined histologically, none showed the brain abnormalities reported by Inman (Figures 4, 5). Talpid^2 limb development, however, is markedly retarded, and digit differentiation is especially slow (Figures 2, 3 and 7).

We prefer not to refer to the presence of supernumerary digits in the talpid^2 phenotype (with inference that nonsupernumerary digits, i.e., normal, are present). In this mutation, unlike diplopodia, polydactyly (*Po*) or duplicate $(Po^4)^5$, the entire footplate is remodeled; no digits of normal shape and constitution are present. Similarly, the wing digits cannot be

TABLE III. Segregation of talpid^2 and diplopodia 1954-1957

Mating type	Numbers of*				Of total identified		Of total embryos
	U	N	D	T	%D	%T	%U
$+dp\ +ta^2 \times +dp\ +ta^2$	58	631	192	269	17.6	24.6	5.0
$+dp\ +ta^2 \times ++\ +ta^2$	37	382	0	122	0	24.2	6.8
$+dp\ +ta^2 \times +dp\ ++$	89	1,610	506	0	23.9	0	4.0
$++\ +ta^2 \times ++\ +ta^2$	2	22	0	6	0	21.4	6.7
$+dp\ ++ \times +dp\ ++$	19	445	129	0	22.5	0	3.2
$+dp\ ++ \times ++\ +ta^2$	4	37	0	0	0	0	9.8
$+dp\ +ta^2 \times ++\ ++$	29	841	0	0	0	0	3.3
$++\ +ta^2 \times ++\ ++$	7	225	0	0	0	0	3.0
$+dp\ ++ \times ++\ ++$	17	667	0	0	0	0	2.5
$++\ ++ \times ++\ ++$	14	224	0	0	0	0	5.9

*U = unidentified early dead; N = normal; D = diplopod; T = talpid.

interpreted as replicate pollices (Figures 1 and 3).

Saunders[8, 9] and particularly Saunders *et al.*[10] have reported that in the chick limb the apical ectodermal ridge exercises an inductive action on subadjacent limb mesoderm to bring about the formation of terminal limb parts. Zwilling[12, 13, 14], studying the mutant "wingless," found that in contrast to normal embryos the apical cap of the mutant wing degenerates and disappears at an early age. He concluded that maintenance of a normal configuration was dependent upon the action of mesodermal factors. Zwilling and Hansborough[15] found that in stages 18 to 20 (Hamburger and Hamilton[4]) polydactylous and normal wing buds neither differed in size nor in the appearance of the apical ectodermal ridge. Increased limb outgrowth in polydactylous embryos was due to the development of an active ectodermal ridge extending further anteriorly than normal. Grafts of the ectodermal covering from limb buds of a normal embryo to the denuded mesodermal core of the limb bud of a polydactylous embryo underwent a pre-axial extension of the ectodermal ridge, this extension corresponding to a pre-axial enlargement of the underlying mesoderm, which preceded the appearance of supernumerary digits. Thus they concluded that the ridge developed the active condition in response to a mesodermal factor, independent of the mass of tissue itself.

These findings led us to examine the apical ectodermal ridge in the limb buds of talpid^2 embryos, which are characterized by a more drastic alteration in limb morphology than other forms of polydactyly. While we have not attempted the careful measurements reported by Zwilling and Hansborough, we have observed an increase in the area of the apical ridge in talpid^2 limbs in contrast to normal limbs of the same age. In the later stages examined we find that the ridge remains thick and "functional" over the digit-forming area of talpid^2 embryos long after its regression in normal siblings (Figure 7). It must be remembered, however, that digit differentiation is considerably delayed in talpid^2 embryos and consequently that stages of equivalent age in

SEPARATED FOOTPLATE

Figure 6

A talpid^2 embryo from a $+dp+ta^2 \times +dp+ta^2$ mating with a footplate separated into two parts. This arrangement may characterize $dpdp\ ta^2ta^2$ individuals. Note spatulate wing with multiple fingers, severe dwarfing, beak and facial abnormalities, celosomia, subcutaneous edema and hemorrhage.

TABLE IV. Sex ratio and age at death of talpid^2 embryos*

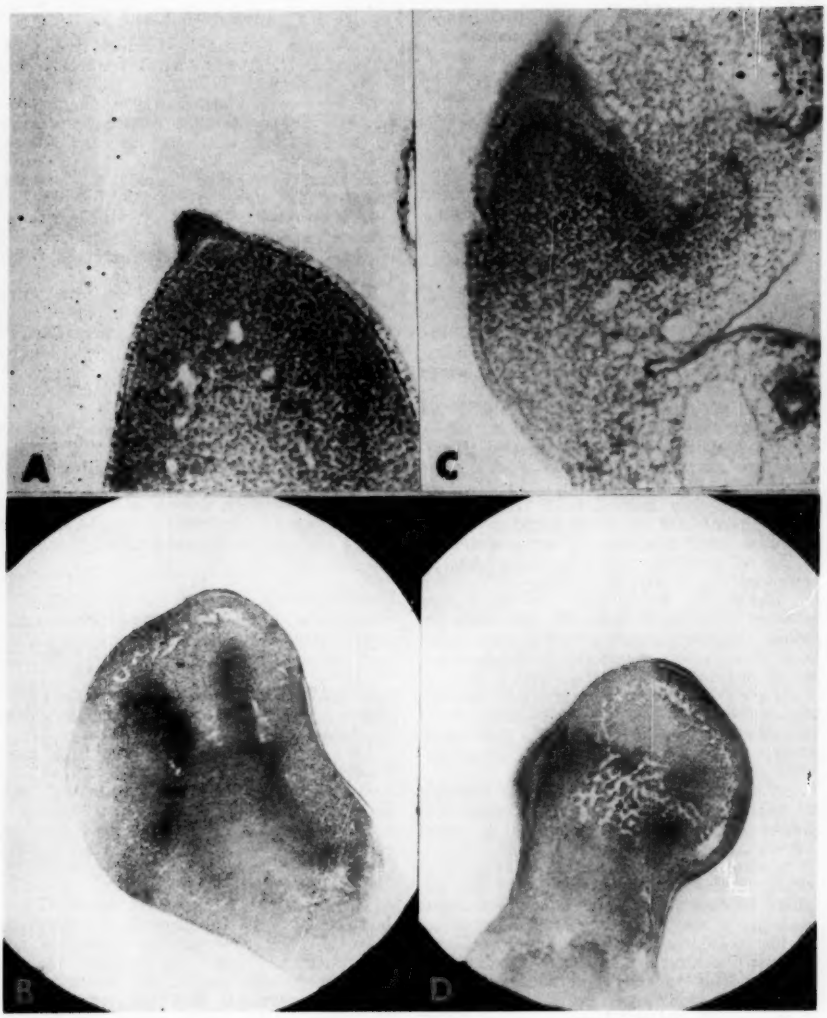
Matings segregating for talpid^2

Sire	Number of talpid^2	♂♂	♀♀	Not classified	Percent ♂♂	Average age at death (days of incubation)
K80	72	28	31	13	47.5	13.1
P124	2	2	—	—	100.0	11.0
R359	13	4	8	1	33.3	12.7
P165	27	11	13	3	45.8	13.3
Total	114	45	52	17	46.4	13.1

Matings segregating for talpid^2 and diplopodia

Sire	Number of talpid^2	♂♂	♀♀	Not classified	Percent ♂♂	Average age at death (days of incubation)
K80	123	50	53	20	48.5	13.3
P124	75	26	40	9	39.4	13.8
R359	24	14	8	2	63.6	12.6
Total	222	90	101	31	47.1	13.4

*1956 and 1957 data.



NORMAL AND TALPID² REAR LIMBS COMPARED

Figure 7

Note the differences in shape, orientation of apical ectodermal ridge and rate of formation of cartilaginous bone models. *A*—Four-day normal embryo (81×). *B*—Five-day normal embryo (30×). *C*—Four-day talpid² embryo (81×). *D*—Five-day talpid² embryo (30×).

time are not equivalent in form.

Our studies of limb differentiation in talpid² embryos do not disagree with the suggested organizer action of the apical ectodermal ridge. We find that the cells present in Saunder's ridge differ in shape and are more abundant in locations corresponding to those of potential digits. Apparently talpid² embryos differ from normal in the ability of the mesodermal core of limb tissue to induce and maintain an apical ectodermal ridge. It is probable, however, that the alteration observed is more

complex than that reported in polydactyly and wingless in that the increase in numbers of digits is accompanied by a remodeling of the entire limb with concomitant distortion in digit form. It is not possible to say whether the maintenance of an active-appearing ectodermal ridge in later stages than in normal embryos reflects only the delay in talpid^2 developmental rate or indicates that the organizer of the apical cap in mutants has a longer period in which to influence the underlying mesoderm.

Summary

An autosomal recessive gene, *ta*^{2}, was detected in a White Leghorn stock selection for shank length. The phenotype, talpid^2, resembles talpid in many respects, including gross and histological structure of the wings and feet and body form. It differs from Inman's histological description of the brain and associated neural structures of talpid, making it quite possible that the two phenotypes are due to different genes. Average age at death of lethal embryos was 13 days. In this respect they differed from Cole's talpid, in which deficient ratios suggested a possible excessive early mortality of homozygotes.

The *ta*^{2} gene was tested in matings into an unrelated line of White Leghorns segregating for diplopodia; the *dp* gene also alters digit number, facial parts, size, etc. The data obtained indicate: (1) that *ta*^{2} is not linked with *dp*; (2) that the double homozygote has the talpid^2 rather than the diplopod phenotype, although a split in the footplate may possibly distinguish the *dpdp* *ta*^{2}*ta*^{2} class from $++ta^2ta^2$ or $+dp\ ta^2ta^2$; and (3) that talpid^2 is expressed fully in the Size $\times$ diplopod genetic background.

Studies of early stages in limb development of talpid^2 and normal embryos revealed that Saunder's presumed limb organizer, the apical ectodermal ridge, is altered in talpid^2 embryos. A general enlargement and some evidence of change in cell shape and orientation may explain the excessive differentiation of terminal limb parts encountered in talpid^2 embryos. Unlike polydactyly where an increase in the area of the anterior portion of the limb organizer leads to the appearance of normal (posterior) and supernumerary (anterior) digits, talpid^2 embryos have no normal digits, suggesting that the entire apical ectodermal ridge may be affected.

Literature Cited

1. Abbott, Ursula K. Further studies on diplopodia. II. Embryological features. *Jour. Genet.* 56:179-196. 1959.

2. ————. Further studies on diplopodia. III. The relationship between expressivity and penetrance in selected lines and crosses. *Jour. Genet.* 56:197-225. 1959.

3. Cole, R. K. The "talpid lethal" in the domestic fowl. *Jour. Hered.* 33:82-86. 1942.

4. Hamburger, V. and H. L. Hamilton. A series of normal stages in the development of the chick embryo. *Jour. Morph.* 88:49-92. 1951.

5. Hutt, F. B. Genetics of the Fowl. McGraw-Hill Book Co. New York. 1949.

6. Inman, Ottilie R. Developmental anomalies in the talpid lethal (*ta ta*) chick. Thesis. Cornell Univ. 110 pp. 1946.

7. Lerner, I. M. Genetic Homeostasis. Oliver and Boyd. Edinburgh. 1954.

8. Saunders, J. W., Jr. The proximo-distal sequence of origin of the parts of the chick wing and the role of the ectoderm. *Jour. Exp. Zool.* 108:363-404. 1948.

9. ————. An analysis of the role of the apical ridge of ectoderm in the development of the limb bud in the chick. *Anat. Rec.* 105: 567-568. 1949.

10. ————, J. M. Cairns and M. T. Gasseling. The role of the apical ridge of ectoderm in the differentiation of the morphological structure and inductive specificity of limb parts in the chick. *Jour. Morph.* 101:57-87. 1957.

11. Taylor, L. W. and C. A. Gunns. Diplopodia: a lethal form of polydactyly in chickens. *Jour. Hered.* 38:66-76. 1947.

12. Zwilling, E. The role of epithelial components in the developmental origin of the "wingless" syndrome of chick embryos. *Jour. Exp. Zool.* 111:175-188. 1949.

13. ————. Reciprocal dependence of ectoderm and mesoderm during chick embryo limb development. *Amer. Nat.* 90:257-265. 1956.

14. ————. Interaction between limb bud ectoderm and mesoderm in the chick embryo. IV. Experiments with a wingless mutant. *Jour. Exp. Zool.* 132:241-253. 1956.

15. ———— and L. A. Hansborough. Interaction between limb bud ectoderm and mesoderm in the chick embryo. III. Experiments with polydactylous limbs. *Jour. Exp. Zool.* 132:219-239. 1956.

CHROMOSOME NUMBERS OF POINSETTIA

Euphorbia pulcherrima Klotzsch

L. C. Ewart and D. E. Walker*

Euphorbia pulcherrima Klotzsch belongs to the Euphorbiaceae, a large and variable family of about 250 genera[2] and between 1000 and 8000 species[1, 8]. *E. pulcherrima*, commonly called poinsettia, was introduced into North America in 1838 from Mexico[6], and since that time it has achieved considerable economic importance as a pot plant crop in the florist trade. Several named types are found in the trade, and in this presentation the term cultivar will be used to denote a selection or variant which has originated or has been maintained only in cultivation. This follows the recommendation of the International Code of Nomenclature for Cultivated Plants[12]. The present day cultivars are presumed to have originated as chance seedlings or to have arisen as somatic mutants[6, 9].

Very limited reports of genetic or cytological studies of poinsettia are found in the literature. The only reported determination of chromosome numbers was made by Moyer[7] on two cultivars, "White" and "Oak" and a seedling resulting from a cross between them, all of which had a somatic number of 28.

Two major objectives were considered in the present investigation. The first objective was to adapt the smear technique of Smith[11], particularly for root tips and pollen-mother-cells. The second objective was to verify reported chromosome counts, to make counts on additional cultivars and hybrids and to observe chromosome behavior and relationships. An investigation of chromosome numbers and relationships would be of value in making a more precise determination of the origin of the cultivars, which in turn would facilitate their use in a breeding program.

Materials and Methods

Nine poinsettia cultivars were studied along with several hybrids and backcrosses and a few lines inbred for one or two generations (Table I). Self-pollinations were made to establish inbred lines. Crosses between the various noninbred cultivars were attempted in order to produce hybrids which were in turn crossed to their respective parents for the backcross generations. All pollinations were performed under controlled conditions in a screened greenhouse. Flowers on the female parents were emasculated daily, since new staminate flowers appear over an extended period, and pollen was applied several times to insure fertilization. One or two seeds were produced by each flower and these required approximately five months to mature. The nearly ripe pods were bagged to contain the seeds as they are forcibly expelled at maturity. Seeds sown in June produced flowering plants in December of the same year.

Chromosome counts were made from root-tip cells in all instances and verified with counts of pollen-mother-cells in some. Immediately after collection the root tips were pretreated for four hours in a saturated solution of paradichlorobenzene to arrest cell division beyond metaphase. After pretreatment, the root tips were killed and fixed from 4 to 24 hours at room temperature in a modified Carnoy's solution consisting of three parts chloroform, two parts 95 percent

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ethyl alcohol and one part glacial acetic acid. After fixation, the tips were hydrolized for 10 minutes at room temperature in a 1:1 solution of concentrated hydrochloric acid and 95 percent ethyl alcohol. After this treatment it was necessary to wash the material very thoroughly in order not to interfere with staining. Removal of the root cap when the material was placed on the slide facilitated smearing. Four stains were tried: aceto-carmine, aceto-orcein, propiono-carmine and propiono-orcein. Aceto-orcein was found to be the best stain for root tips in that it stained the chromosomes and left the cytoplasm clear. The pollen-mother-cells were killed and fixed in the modified Carnoy's solution and smeared in aceto-carmine with no other treatments necessary before smearing and staining.

Prepared slides of both root tips and pollen-mother-cells sealed with a mixture of gum mastic and paraffin stored very well for several weeks when placed in the hydrator of a laboratory refrigerator.

The time of day for collecting the root tips was very important. Temperature was probably a factor in this seasonal variation of time of collection. By far the maximum number of dividing cells were found in material collected about 9 a.m. in the spring months and 1 p.m. in the winter. These times agreed closely with those reported by Hill and Carnahan[4] and Rogers[10]. Photomicrographs were made at a magnification of 970×, and photodrawings prepared according to the procedure described by Manton[5] were found to be preferable to camera lucida drawings.

Results

Chromosome numbers of nine noninbred cultivars and several cultivars inbred for one or two generations, along with F_1, F_2 and backcross hybrids, are given in Table I. Without exception, the somatic numbers determined from counts of 10 to 22 root-tip cells from one to five plants were found to be either 28 or 56. Four cultivars, "Barbara Ecke Supreme," "Improved Albert Ecke," "Mrs. Paul Ecke" and "Indianapolis Red," had 56 as their $2n$ number, while five, "Ruth Ecke," "Oakleaf," "St. Louis," "White Ecke" and "Henrietta Ecke," displayed a somatic number of 28. "Mrs. Paul Ecke" ($2n=56$) and "White Ecke" ($2n=28$) are shown in Figure 8*A-D*.

S_1 generation progeny of five cultivars and S_2 generation progeny of one cultivar were grown and counts of root-tip cells were made (Table I). There were no deviations from those found in the respective noninbred parents, even in the 56-chromosome types.

F_1 and F_2 hybrids from crosses between cultivars with a somatic number of 28 displayed the expected number of chromosomes in all instances (Table I). However, crosses between cultivars with different chromosome numbers gave unexpected results. "St. Louis" (28) × "Improved Albert Ecke" (56), "St. Louis" (28) × "Barbara Ecke Supreme" (56) and "White Ecke" (28) × "Barbara Ecke Supreme" (56) all showed $2n=28$, while "Oakleaf" (28) × "Barbara Ecke Supreme" (56) showed $2n=56$. In no case was the expected $2n$ number of 42 observed. Twelve progeny were grown in the first three crosses and three in the last. No seeds were produced when a 56-chromosome type was used as a female parent except as a result of self-pollination.

Backcrosses involving only 28-chromosome cultivars showed no irregularity in chromosome number (Table I). Only one backcross was studied in which a 56-chromosome cultivar was in the parentage, namely, "Oakleaf" × "Barbara Ecke Supreme" backcrossed to "Oakleaf" in which the five progeny showed $2n=28$. The chromosome number of the F_1 plant used in this backcross was not determined because the plant died before the counts were made. Therefore, it is possible that the F_1 used in the backcross had a chromosome number of 28. If so, the backcross progeny would have been expected to exhibit a somatic number of 28. Lack of inbred parents and a small number of progeny from a cross precluded genetic analysis. However, the observation of a recombination of bract

color and leaf shape is evidence that hybridization had taken place.

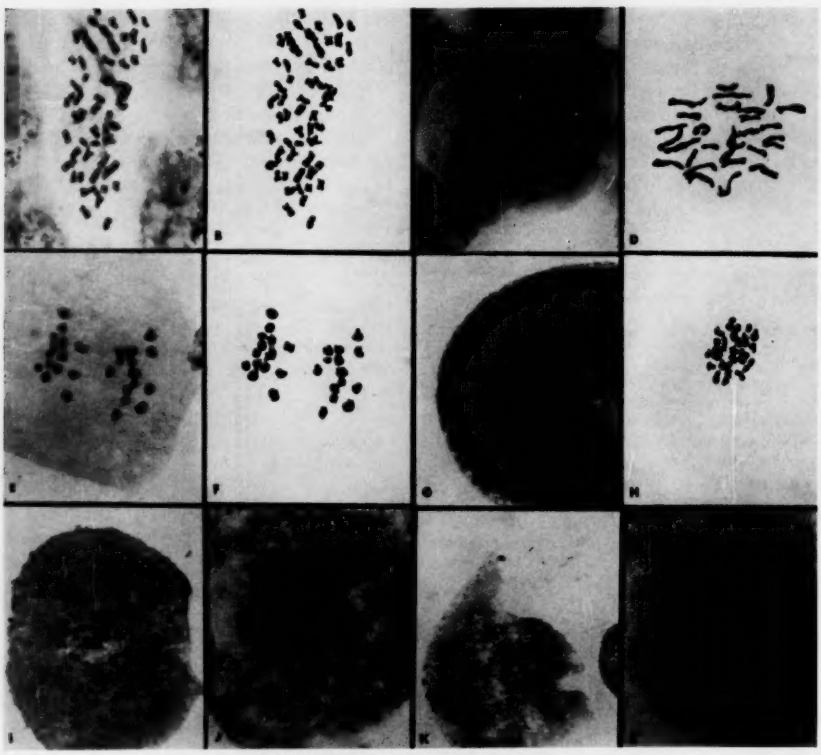
Because these unusual results were observed in the crosses between cultivars of different chromosome numbers, examinations were made of pollen-mother-cells of five of the noninbred cultivars to see if any meiotic irregularities were occurring. Regular disjunction occurred in both the 28- and 56-chromosome types (Figure 8*E-H*). Also, the *n* numbers of chromosomes given in the last column of Table I agree with those found from counts of root tips.

Meiotic cells showed further that four or more chromosomes were associated not only in the 56-chromosome types (Figures 8*I* and *J*) but also in some of

TABLE I. Chromosome numbers of cultivars, inbreds and hybrids of poinsettias

Cultivars, inbreds and hybrids	2*n* no. of parents	No. of plants	Root tips No. cells counted	Root tips 2*n*	PMC
Cultivars:					
Barbara Ecke Supreme (BES)[1]	5	—	17	56	28
Improved Albert Ecke (IAE)	—	—	13	56	28
Mrs. Paul Ecke (MPE)	—	—	10	56	—
Indianapolis Red (IR)	—	—	10	56	—
Ruth Ecke (RE)	—	—	22	28	14
Oakleaf (OL)	—	—	10	28	14
St. Louis (St.L)	—	—	10	28	—
White Ecke (WE)	—	—	21	28	14
Henrietta Ecke (HE)	—	—	10	28	—
Inbreds (S_1)[2]:					
BES	56	5	15	56	—
IR	56	3	10	56	—
IAE	56	3	10	56	—
OL	28	1	10	28	—
RE	28	1	10	28	—
Inbreds (S_2)[2]:					
IAE	56	5	10	56	—
Hybrids (F_1)[3]:					
OL × WE	28 × 28	3	12	28	—
HE × WE	28 × 28	2	10	28	—
WE × St.L	28 × 28	3	10	28	—
St.L × WE	28 × 28	4	10	28	—
St.L × IAE	28 × 56	5	11	28[6]	—
St.L × BES	28 × 56	4	11	28[6]	—
WE × BES	28 × 56	3	10	28[6]	—
OL × BES	28 × 56	3	10	56[6]	—
Hybrids (F_2):					
WE × RE	28 × 28	5	14	28	—
Hybrids (BC)[4]:					
(OL × BES) × OL	(28 × 56) × 28	5	10	28[6]	—
(RE × WE) × RE	(28 × 28) × 28	4	10	28	—
(WE × RE) × RE	(28 × 28) × 28	5	12	28	—
(WE × RE) × WE	(28 × 28) × 28	5	10	28	—
(WE × OL) × OL	(28 × 28) × 28	3	15	28	—
(WE × OL) × WE	(28 × 28) × 28	4	10	28	—

[1]Abbreviations in parentheses after cultivar name used in remainder of table.
[2]S_1 and S_2 represent one and two generations of selfing.
[3]F_1 is a hybrid between noninbred cultivars.
[4]BC is an F_1 backcrossed to a noninbred cultivar.
[5]No count made or column not appropriate.
[6]Unexpected numbers from parents of differing numbers of chromosomes.



POINSETTIA CHROMOSOMES

Figure 8

A—Root-tip cell of "Mrs. Paul Ecke," $2n=56$. *B*—Photodrawing of *A*. *C*—Root-tip cell of "White Ecke," $2n=28$. *D*—Photodrawing of *C*. *E*—Anaphase I of "Oakleaf," $n=14$. *F*—Photodrawing of *E* showing association of chromosomes and regular disjunction. *G*—Anaphase II of "Barbara Ecke Supreme" showing regular disjunction, $n=28$. *H*—Photodrawing of part of *G*. *I*—Anaphase I of "Improved Albert Ecke" showing multivalents, $n=28$. *J*—Diakinesis of "Indianapolis Red" showing multivalents, $2n=56$. *K*—Diakinesis of "St. Louis" showing multivalents, $2n=28$. *L*—Diakinesis of "Oakleaf" showing multivalents, $2n=28$. (The authors wish to thank Miss Ok Joo Pai for supplying photomicrographs of *I*, *J* and *K*.)

the 28-chromosome cultivars. In divisions of "Oakleaf," "St. Louis" and "Ruth Ecke" there appear to be several multivalents. Figure 8*K* shows the apparent presence of several multivalents at diakinesis in "St. Louis" and Figure 8*L* shows a similar occurrence in "Oakleaf." Associations of chromosomes were observed in several anaphase I preparations, suggesting that bivalents were going to the poles following multivalent formation in the earlier stages of meiosis.

Discussion

Two questions evolve from these results. The first has to do with the absence of $2n=42$ (triploid) types in the hybrids and the second is in regard to the basic chromosome number of the species.

The nine cultivars fall into two chromosome groups with somatic numbers of 28 and 56. The numbers remained constant after one or two generations of inbreeding and, in the diploids, no deviations were found in the F_1 hybrids and backcrosses. Thus it might have been expected that crosses between 28- and 56-chromosome types would yield triploid ($2n=42$) progeny. Fifteen plants from four such

crosses gave 12 plants with a somatic number of 28, three plants with 56 chromosomes, and none with 42 chromosomes. Remembering that no progeny resulted from crosses in which the tetraploid types were the female parents, several possibilities might be mentioned to account for these results, none of which is entirely satisfactory.

Apomixis would result in progeny with 28 chromosomes but would not account for the segregation and recombinations of leaf shapes and bract color which were observed. Apomixis has been reported in the Euphorbiaceae, e.g., *Euphorbia dulca* and *Alchornea ilicifolia*. They exhibit adventitious embryogeny in which the embryos develop directly from sporophytic tissue (Stebbins[13]). Stebbins further notes that in certain apomicts, some ovules produce diploid embryo sacs through failure of meiosis while other ovules in the same plant yield haploid embryo sacs and egg cells and require fertilization. No mention is made of adventitious embryogeny occurring along with normal sexual reproduction. Meiosis appears to be normal in this case and it seems unlikely that apomixis is responsible for these results. Apomixis alone could not explain the occurrence of a 56-chromosome type from such a cross.

A second cause could be through either nonreduction or normal reduction followed by doubling, with fertilization by diploid pollen resulting in a somatic number of 56. Double reduction of the pollen-mother-cells would be necessary to account for the 28-chromosome progeny. Further investigations are necessary to resolve this problem. No one mechanism explains the appearance of both chromosome numbers from the same type of cross.

More evidence is available to answer the second question concerning the basic chromosome number of the species. The observations suggest that the basic number is 7 rather than 14, as might be inferred from the constancy of somatic numbers of 28 and 56 of the cultivars. If the polyploid cultivars originated as mutants of the diploids and the basic number is 14, only bivalents would be expected in the diploids. Multivalents in diakinesis and bivalents moving toward the poles in anaphase I found in the 28-chromosome types suggest that more than two homologues are present in the nucleus. These aberrant bivalents could either result from earlier quadrivalent association or precocious separation during the first division. The observations favor the first of these two explanations.

The 56-chromosome cultivars have originated relatively recently and apparently due to chromosome doubling. It might be reasonably assumed that some time in the past this had occurred once before, giving rise to a somatic number of 28 from a 14-chromosome progenitor. A basic number of 7 for this species would be in agreement with that reported for *Euphorbia bivanae, E. pubescens* and *E. dentata*[3].

A more complete analysis of chromosome morphology and behavior will be necessary to produce more conclusive reasons for the absence of triploid hybrids as well as a more definitive answer to the question of the basic chromosome number for the species. The discovery of a wild species with a somatic number of 14 would, of course, be the best evidence for a basic number of 7.

Summary

Smear preparations proved to be satisfactory for making studies of the chromosomes of cultivated poinsettias. Staining with aceto-orcein gave best results for root tips while aceto-carmine was most satisfactory for pollen-mother-cells. Of the nine cultivars studied, five had somatic numbers of 28 and four had 56. Twenty-eight-chromosome types selfed, crossed and backcrossed, and 56-chromosome types selfed gave the expected numbers in the progeny. In crosses of 28 × 56-chromosome types, only progeny with 28 or 56 chromosomes were observed and none with 42. The data suggest no satisfactory explanation for the absence of 42-chromosome progenies. Plants having 56 chromosomes were unsuccessful as female parents except when selfed. Meiotic studies of several cultivars confirmed the somatic counts. The chromosomes showed regular disjunction and, in addition, displayed multivalents in both the 28- and 56-chromosome cultivars. Multivalent formation in the 28-chromosome cultivars suggests that they may be polyploids and that the basic number for the species is 7.

Literature Cited

1. Bailey, L. H. The Standard Cyclopedia of Horticulture. MacMillan Co. Chicago. Vol. 1. pp. 1167-1174. 1947.

2. Cummins, G. B., S. N. Postlethwait, G. A. Gries and F. W. Stearns. Textbook of Intermediate Plant Science. Burgess Co. Minneapolis. p. 29. 1950.

3. Darlington, C. D. and Ann P. Wylie. Chromosome Atlas of Flowering Plants. MacMillan Co. New York. pp. 126-128. 1956.

4. Hill, Helen and H. L. Carnahan. Effects of variables in weather and their interactions on metaphase cells in root tips of grasses. *Bot. Gaz.* 116:82-85. 1954.

5. Manton, Irene. Problems of Cytology and Evolution in the Pteridophyta. Cambridge University Press. pp. 298-299. 1950.

6. Moon, Mary H. A short history of the poinsettia. *Baileya* 4:176-180. 1956.

7. Moyer, L. S. Electrophoresis of latex and chromosome numbers of poinsettias. *Bot. Gaz.* 95:678-685. 1934.

8. Percy-Lancaster, S. Poinsettias. *Gard. Chron.* 97:76. 1935.

9. Post, K. Florist Crop Production and Marketing. Orange Judd Co. New York. pp. 494-509. 1959.

10. Rogers, O. M. Cytological studies and genetic relationships in the Gesneriaceae. M.S. Thesis. Cornell University. 34 pp. 1954.

11. Smith, L. The aceto-carmine smear technic. *Stain Tech.* 22:17-31. 1947.

12. Stearn, W. T. International Code of Nomenclature for Cultivated Plants. Royal Horticultural Society. London. 29 pp. 1953.

13. Stebbins, G. L. Variation and Evolution in Plants. Columbia University Press. 1950.

AN ALTERNATIVE EXPLANATION FOR SEX REVERSAL AND ABNORMAL SEX RATIOS IN THE FOWL

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In a recent paper in the *Journal of Heredity*, Carne and Waterhouse[1] described a case of sex reversal and abnormal sex ratio in the fowl. A sex-reversed female sired progenies which, though few survived hatching, were all male. In their explanation, the authors assumed that the sex reversal was due to the gain of an X chromosome by the female, thus causing the bird to become a genetic male. They concluded that, "It is difficult in the absence of further evidence to put forward an explanation which carries any conviction. If we suppose that the absence of females is due to both X chromosomes of the cock carrying a lethal, we must further suppose that the two lethal genes are closely linked or included in a pericentric inversion, for which the cock is heterozygous, otherwise crossing-over would result in some X chromosomes carrying no lethal and none were found." Further, "To account for the lethal effect in males the genes must be supposed dominant and this makes it very hard to account for the cock himself . . ." They suggested the possibility of a translocation between the X chromosomes occurring at an early cleavage division, thus providing the cock with a complete genome but resulting in unbalanced gametes which would be lethal in embryogeny.

Recent work in our laboratory suggests an alternative theory for the explanation of the sex reversal and abnormal sex ratios. It was found that a genetic female became a phenotypic and functional male, at least to the extent of its gonads functioning in spermatogenesis[2], as a result of polysomy not involving the sex chromosome. This suggests an X/autosomal balance mechanism for sex determination in the fowl and may offer a simpler explanation for the abnormal sex ratio observed by Carne and Waterhouse.

If the fowl in question carried only one X chromosome and was polysomic, only one kind of sperm would be produced, i.e., those carrying an X chromosome and extra autosomes (those deficient for the X chromosome, even though polysomic, being gametically lethal). Thus any polysomic X-carrying sperm fertilizing any egg with or without an X chromosome would produce a male. The zygotes carrying two X chromosomes would be genetic males, and those carrying one X but polysomic would also be phenotypically males though genetically females by their X chromosome constitution. The lethality in early embryogeny may be explicable as a consequence of polysomy interfering with normal genetic development at critical periods of embryogenesis.

The process may be diagrammed as follows:

P_1 sex-reversed female functioning as male $\times$ normal female

$A^aA^aXO \times AAXO$

P_1 gametes: A^aX, $A^aO \times AX$, AO

F_1 A^aAXX = unbalanced male
A^aAXO = genetic female but phenotypic male
(A^aAOO) = deficient for X, presumed gametic lethal

(superscript a = extra autosomes)

This seems to be in line with experimental evidence and offers a reasonable explanation for the data of Carne and Waterhouse if we grant the assumption that their sex-reversed female was induced by an unbalanced X/autosomal condition. Since either infection or incipient neoplasia can produce polysomy, cytological examination of the gonads would be necessary for proof.

Literature Cited

1. Carne, H. R. and W. L. Waterhouse. Sex-reversal and abnormal sex ratios in the domestic fowl. *Jour. Hered.* 49:102, 106. 1958.

2. Newcomer, E. H., Grace M. Donnelly and Wm. C. Forbes. A cytological study of sex reversal in the fowl. *Poultry Sci.* 39:1030-1037. 1960.

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THE INHERITANCE OF MOTTLED LEAF IN COTTON

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THE Texas Agricultural Experiment Station maintains a large collection of species of *Gossypium*, races of *G. hirsutum* and genetically marked stocks. Among these cottons is an introduction of *G. hirsutum* L. with a peculiar light green mottling of the leaf blades. This strain was sent to C. B. Doyle from French Equatorial Africa in 1948 and was designated CB 2046. It was listed as Texas 92 in the catalogue of *Gossypium* stocks maintained under Regional Project S-1, Genetics and Cytology of Cotton.

The light colored areas occur at random over the leaves, and vary in size and shape. Individual areas are usually one to two millimeters square, but larger patches of light green tissue are often found. There is much more normal green tissue than light green in a mottled leaf. The character is not conspicuous and colorful but can be detected and scored with confidence. The mottled areas can be seen clearly when sunlight shines through the leaf.

MOTTLED LEAF

Figure 9

The lighter colored areas scattered over the leaf and concentrated along the main veins are characteristic of this mutant.

Review of the Literature

Phenomena such as this mottled leaf character have been described in the literature as mottled, mosaic and variegated. Lewis[4] reported a mutant causing a mosaic leaf in cotton which was inherited as a recessive. The character called mottled leaf is not nearly so conspicuous and colorful as mosaic leaf, although both traits apparently are cases of somatic instability. As stated in the earlier paper, individual cases of variegated inheritance have been attributed to mutable genes, unstable interaction between nonallelic genes, varie-gated-type position effect and unstable plastid development. Specific references to these cases are cited in the discussion.

Materials and Methods

The mottled leaf parent used in this experiment was *Gossypium hirsutum* stock CB 2046 catalogued as Texas 92, and the nonmottled or normal green stock was a selfed line of Deltapine 14 maintained at the Texas Station as a genetic stock. Appropriate crosses and selfs were made to furnish seed for small populations of parental, F_1, F_2 and backcross populations in 1956. When it was learned that plants in the segregating populations could be scored with certainty, seeds were produced for larger

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populations in 1957. Since the results of the limited planting in 1956 suggested that a recessive gene was involved in the inheritance of mottled leaf, segregants from the F_2 and backcross populations were progeny tested in 1957 to confirm this hypothesis.

All seeds were planted individually in six-ounce paper cups in the greenhouse and in approximately two weeks the seedlings were transplanted to the field at 24-inch spacing in rows 40 inches wide. No attempt was made to classify the plants until they had reached maturity. For economy of space in the tables, Texas 92 and Deltapine 14 are abbreviated 92 and D & PL, respectively.

Results

The classification of plants in the parental, F_1, F_2 and backcross populations involving Texas 92 and Deltapine 14 is recorded in Table I. All Texas 92 plants developed the characteristic mottled leaf appearance. Over the years hundreds of plants of the selfed line of Deltapine 14 had been grown and all had normal green leaves. The F_1 Deltapine 14 × Texas 92 showed no trace of the mottled leaf character. The F_2 Deltapine 14 × Texas 92 segregated into 3 nonmottled to 1 mottled leaf. The backcross of (F_1 Deltapine 14 × Texas 92) × Texas 92 segregated into a 1:1 ratio. The backcross of (F_1 Deltapine 14 × Texas 92) × Deltapine 14 contained all nonmottled plants.

The data indicated that the mottled leaf expression was controlled by one locus, with mottled leaf recessive to normal green. In accordance with rules for symbolizing genes in cotton, the symbol *mt* is assigned to this mutant. It was assumed that Texas 92 had the genotype *mtmt*, and Deltapine 14 *MtMt*.

Seeds from plants from the 1956 F_2 and backcross populations were available, and the results of the progeny tests of 13 plants are recorded in Table II. Three F_2 plants with the mottled leaf phenotype were progeny tested. They were assumed to have the *mtmt* genotype, and all plants in the three progenies developed the mottled leaf expression.

Nonmottled leaf plants in the F_2 could either have been *MtMt* or *Mtmt*. Three plants were progeny tested; two progenies segregated into a 3:1 ratio of nonmottled to mottled leaf and were considered to have the *Mtmt* genotype. The other progeny had only nonmottled leaf plants and was considered to be *MtMt*.

According to the hypothesis, nonmottled leaf segregants from the backcross (F_1 Deltapine 14 × Texas 92) × Texas 92 would have the genotype *Mtmt*, and

TABLE I. Classification of plants in parental, F_1, F_2 and backcross populations involving Texas 92 and Deltapine 14

Progeny	No. of plants nonmottled	No. of plants mottled	Chi-square analysis
Texas 92	0	14	
D & PL 14	15	0	
F_1 D & PL 14 × 92	15	0	
F_2 D & PL 14 × 92 (1956)	31	14	0.90 (3:1) p = .30 — .50
" " × " (1957)	190	68	0.25 " p = .50 — .95
Pooled F_2	221	82	0.69 " p = .30 — .50
(F_1 D & PL × 92) × 92 (1956)	18	27	1.80 (1:1) p = .10 — .20
(" " × ") × 92 (1957)	32	33	0.02 " p = .50 — .95
Pooled backcross	50	60	0.91 " p = .30 — .50
(F_1 D & PL × 92) × D & PL (1956)	15	0	
(" " × ") × D & PL (1957)	14	0	
Pooled backcross	29	0	

such plants should segregate into a 3:1 ratio. Two plants were progeny tested, and the classification of the plants fit the theoretical ratio.

Plants from the backcross (F_1 Deltapine 14 × Texas 92) × Texas 92 were all nonmottled leaf, but two genotypes, *MtMt* and *Mtmt*, were expected in equal numbers. Five plants from this population were progeny tested, but all five proved to be *MtMt* for no mottled leaf plants appeared.

Discussion

The data showed that the mottled leaf expression was controlled by one gene pair, with mottled leaf recessive to normal green. This explanation of the inheritance of a variegated leaf in cotton was similar to the report of mosaic leaf inheritance by Lewis[4], although the two traits were distinctly different in outward appearance. The genetic results in each case indicated that a stable recessive gene was involved, and there was no evidence for mutable loci as reported by Emerson[2], Jones[3], Smith and Sands[8] and others. Since the mottled leaf study involved an intraspecific cross of two *G. hirsutum* stocks with normal chromosome pairing, there was no reason to suspect variegated-type position effects as reported by McClintock[5, 6]. Somatic reduction, as reported by Brown[1] and Menzel[7], was not a likely possibility. The two cases in cotton were similar to the case of variegated leaf in *Nepeta* reported by Woods and DuBuy[9]. The homozygous recessive condition allowed changes to occur in plastid development in certain cells, which resulted in discolored areas on the leaf. The changes did not occur in all cells, and that is the inexplicable feature of cases of variegated inheritance which are not accounted for by mutable genes. As pointed out by Smith and Sands[8], the problem of variegation is fundamentally the question of how any cells or tissues differentiate, whether normal or abnormal. Normal differentiation seemingly is an orderly process governed by the genotype of the organism, but variegation occurs in an erratic manner. The mechanism for the internal changes which result in the discolored areas on the leaf has not been determined.

Summary

A mutant designated as mottled leaf arose in the progeny of a *Gossypium hirsutum* introduction. Genetic studies of the inheritance of this trait showed that it was controlled by one gene pair, with mottled leaf recessive to normal green. *Mt* was assigned as a symbol for this gene.

Literature Cited

1. Brown, Meta S. A case of spontaneous reduction of chromosome number in somatic tissue of cotton. *Amer. Jour. Bot.* 34:384-388. 1947.

2. Emerson, R. A. The inheritance of a recurring somatic variation in variegated ears

TABLE II. Classification of plants in progeny tests of F_2 and backcross segregants involving mottled leaf and Deltapine 14

Plant no.	Progeny	Assumed genotype	No. of plants nonmottled	No. of plants mottled	Chi-square analysis
1	F_3 D & PL × Texas 92	*mtmt*	0	30	
2	F_3 " × "	" "	0	15	
3	F_3 " × "	" "	0	15	
			0	60	
4	F_3 D & PL × Texas 92	*Mtmt*	25	5	1.11 (3:1) p = .20 — .30
5	F_3 " × "	" "	22	8	0.04 " p = .50 — .95
	Pool seg. F_3	" "	47	13	0.36 " p = .50 — .95
6	F_3 D & PL × Texas 92	*MtMt*	30	0	
7	F_2 of (F_1 D & PL × 92) × 92	*Mtmt*	24	5	0.93 (3:1) p = .30 — .50
8	F_2 of (" " × 92) × 92	" "	17	9	1.28 " p = .20 — .30
	Pooled	" "	41	14	0.01 " p = .50 — .95
9	F_2 of (F_1 D & PL × 92) × D & PL	*MtMt*	25	0	
10	F_2 of (" " × 92) × "	" "	30	0	
11	F_2 of (" " × 92) × "	" "	28	0	
12	F_2 of (" " × 92) × "	" "	30	0	
13	F_2 of (" " × 92) × "	" "	30	0	
	Pooled		143	0	

of maize. *Amer. Nat.* 48:87-115. 1914.

3. Jones, D. F. Mutation rate in somatic cells of maize. *Proc. Nat. Acad. Sci.* 22:645-648. 1936.

4. Lewis, C. F. Genetic studies of a mosaic leaf mutant causing somatic instability in cotton. *Jour. Hered.* 49:267-271. 1958.

5. McClintock, B. Chromosome organization and genic expression. *Cold Spring Harbor Symp. Quant. Biol.* 16:13-37. 1951.

6. ————. Intranuclear systems controlling gene action and mutation. *Brookhaven Symp. Biol.* 8:58-74. 1956.

7. Menzel, Margaret Y. Polygenomic hybrids in *Gossypium*. III. Somatic reduction in a phenotypically-altered branch of a three-species hexaploid. *Amer. Jour. Bot.* 39:625-633. 1952.

8. Smith, H. H. and S. A. Sand. Genetic studies on somatic instability in culture derived from hybrids between *Nicotiana langsdorffii* and *N. sanderae*. *Genetics* 42:560-582. 1957.

9. Woods, M. W. and H. G. DuBuy. Hereditary and pathogenic nature of mutant mitochondria in *Nepeta*. *Jour. Nat. Cancer Inst.* 11:1105-1151. 1951.

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NOTES ON A STAMINATE GRAPE SELECTION AS A PARENT

N. H. Loomis*

GENETIC information on inheritance of characters in grapes is meager, and records which involve crosses with staminate plants are few. The purpose of this paper is to record information on the progeny of a pistillate-flowered selection when crossed with the staminate rootstock selection Aramon × Rupestris Ganzin No. 1 (hereinafter abbreviated ARG 1).

Materials and Methods

A seedling (Meridian Selection No. 10) with pistillate flowers (reflex stamens) which originated as a cross of Champanel × Diamond was pollinated by ARG 1. Progenies of 78 plants survived to flower and were classified for flower type and fruitfulness.

Results and Discussion

Among 78 seedlings, 38 were perfect-flowered (hermaphroditic) and 40 were staminate. This is a close fit to a normal 1:1 Mendelian ratio, yet none of the progeny was of the same flower type as the seed parent (pistillate).

Oberle[4] developed a hypothesis on the inheritance of flower type which seemed to fit his data, but he did not have sufficient data from staminate-type plants to verify all of it. The result obtained from pollen of the staminate selection ARG 1 fits Oberle's hypothesis and substantiates a portion of the hypothesis not previously verified. Oberle's hypothesis is that the flower type of bunch grapes is controlled by two genes closely linked on the same, chromosome. These genes are indicated by the following symbols:

So—a dominant gene suppressing the development of normal ovules

Sp—a dominant gene for development of normal pollen

The different genetic flower types are.

Pistillate

sosp/sosp

Staminate

Normal wild type *SoSp/sosp*

Derived from a hermaphroditic plant *SoSp/soSp*

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Hermaphrodite

Heterozygous type	*soSp/sosp*
Homozygous type	*soSp/soSp*

Crossing hermaphrodites (either heterozygous or homozygous) with wild-type staminate plants gives some staminate plants which are homozygous for the development of normal pollen. They have the genotype *SoSp/soSp*. ARG 1 is apparently of this type. It originated from seed of the hermaphroditic variety Aramon and pollen of the staminate selection Rupestris Ganzin, which has the "normal wild" genotype.

According to the data obtained in the progeny of Meridian No. 10 × ARG 1, ARG 1 must be homozygous dominant for the development of normal pollen and heterozygous for the factor which suppresses the development of normal ovules, thus *SoSp/soSp*. Therefore, all seedlings will produce normal pollen; half of these will produce normal ovules and be hermaphrodites, and half will not have functional ovules and will be staminate. Crosses of ARG 1 with either pistillate or hermaphroditic selections will always give progeny ratios of 1 hermaphrodite: 1 staminate-type plant.

The French authority P. Galet[2] writes that the Seibel selections 42, 60, 63, 85, 127, 175, 405, 750, 753, 1000, 2041, and 2044 are seedlings of ARG 1, and that Seibel numbers 52 and 2045 probably are, also.

Notes on the flower type of the French hybrids are almost nonexistent. Galet[2] states that all the above Seibel selections are probably hermaphrodites. Numbers 42 and 1000 are known from observation to be, and seven of the others are reported[1,5,6] to be the pollen parents of later selections, so they, too, must be hermaphrodites. No record has been found to indicate that pollen of the staminate grape selection ARG 1 has ever produced seedlings with pistillate-type flowers.

Gladwin[3] used pollen of ARG 1 to: (1) improve the fruit set of imperfectly pollinating and self-sterile varieties which set poor fruit clusters, and (2) breed new grape varieties. He reports that all seedlings of ARG 1 were male plants.

The seed parents used by Gladwin were different from those used in the present study and may have given him an entirely different result. Staminate-type grape vines tend to flower at an earlier age than the fruit-producing types. It is likely that, when Gladwin saw numerous staminate plants among his seedlings and no fruit-producing plants, he destroyed all the vines without waiting for complete progeny records.

It is apparent from the data presented in this paper and from the parentage of Seibel's seedlings that ARG 1 produces some fruitful progeny. It cannot be homozygous dominant for the two genes which control flower type, as Oberle[4] suggested after studying Gladwin's[3] results.

Summary

A pistillate-type grape (reflex stamens) selection when crossed with the staminate selection Aramon × Rupestris Ganzin No. 1 gave a progeny of hermaphroditic and staminate plants in a 1:1 ratio. None of the seedlings had the pistillate-type flowers of the seed parent. Aramon × Rupestris Ganzin No 1 is apparently homozygous for the dominant gene which allows the development of normal pollen, and heterozygous for the gene which suppresses the development of normal ovules. It is the first staminate-type grape plant known to be of this genetic type.

Literature Cited

1. Barrett, H. C. Personal correspondence. November 14, 1958.

2. Galet, P. Personal correspondence. November 24, 1959. December 11, 1959.

3. Gladwin, F. E. Pollination with particular reference to the grape. *Amer. Fruit Grower* 57(3):9, 24-25, and 57(4):16, 35. 1937.

4. Oberle, George D. A genetic study of variations in floral morphology and function in cultivated forms of *Vitis*. *N. Y. Agr. Exp. Sta. Tech. Bul.* 250. 1938.

5. Pee-Laby. La Vigne Nouvelle. Bailliere. Paris. 1959.

6. Perold, A. I. A Treatise on Viticulture. MacMillan and Co. London. 1927.

A PROPOSED STANDARD SYSTEM OF NOMENCLATURE OF HUMAN MITOTIC CHROMOSOMES

THE HUMAN CHROMOSOME STUDY GROUP*

THE rapid growth of knowledge of human chromosomes in several laboratories, following advances in technical methods, has given rise to several systems by which the chromosomes are named. This has led to confusion in the literature and so to the need for resolving the differences. Consequently, at the suggestion of Dr. C. E. Ford, a small study group was convened to attempt to formulate a common system of nomenclature. The meeting was arranged, through the good offices of Dr. T. T. Puck, to be held at Denver, in the University of Colorado, under the auspices of the Medical School. The meeting of this study group was made possible by the support of the American Cancer Society, to whom grateful thanks are due.

For practical reasons, it was decided to keep the group as small as possible and to limit it to those human cytologists who had already published karyotypes.† In addition, three counselors were invited to join the group to guide and aid the discussions and, if necessary, to arbitrate. Fortunately, the last office did not prove necessary, and it was possible by mutual agreement to arrive at a common system which has flexibility.

It was agreed that the principles to be observed by the system should be simplicity and freedom, as far as possible, from ambiguity and confusion, especially with other systems of nomenclature in human genetics. It should also be capable of adjustment and expansion to meet the needs of new knowledge of human chromosomes. The system should be agreed upon by the greatest possible proportion of cytologists working in the field, but the risk that a minority may be unable to accept the system as a whole should not be allowed to delay adoption by a majority.

It was agreed that the autosomes should be serially numbered, 1 to 22, as nearly as possible in descending order of length, consistent with operational conveniences of identification by other cri-

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†In contemporary publications the terms, karyotype and idiogram, have often been used indiscriminately. We would recommend that the term karyotype be applied to a systematized array of the chromosomes of a single cell prepared either by drawing or by photography, with the extension in meaning that the chromosomes of a single cell can typify the chromosomes of an individual or even a species. The term idiogram would then be reserved for the diagrammatic representation of a karyotype, which may be based on measurements of the chromosomes in several or many cells.

teria. The sex chromosomes should continue to be referred to as X and Y, rather than by a number, which would be an additional and, ultimately, a superfluous appellation.

It was generally agreed that the 22 autosomes can be classified into seven groups, distinction between which can readily be made. Within these groups, further identification of individual chromosomes can in many cases be made with relative ease. Within some groups, especially the group of chromosomes numbered 6 to 12, including also the X chromosome, the distinctions between the chromosomes are very difficult to make by presently available criteria. However, lesser difficulties are encountered in separating chromosomes 6 and X from the remainder of this group. It is believed that, with very favorable preparations, distinction can be made between most, if not all, chromosomes.

It is proposed that the autosomes first be ordered by placing the seven groups as nearly as possible in descending order of size. Within each group the chromosomes are arranged, for the most part, by size. It was desired specifically to avoid the implication that size relationships have been permanently decided in every instance, but it is hoped that the assignment of numbers will be permanently fixed. In those cases in which distinction is at present doubtful, final definition of each chromosome can be left until further knowledge has accrued, although an attempt is made to provide a characterization of each. These principles make it possible to draw up a conspectus of the chromosomes, a table of their quantitative characteristics and a table of the synonyms which authors have already published (Tables I, II and III).

In Table II, showing the diagnostic characters of the chromosomes, three parameters are relied upon. These are: (1) the length of each chromosome relative to the total length of a normal, X-containing, haploid set, i.e., the sum of the lengths of the 22 autosomes and of the X chromosome, expressed per thousand; (2) the arm ratio of the chromosomes expressed as the length of the longer arm relative to the shorter one; and (3) the centromeric index expressed as the ratio of the length of the shorter arm to the whole length of the chromosome. The two latter indices are, of course, related algebraically quite simply, but it is thought useful to present both here. In some chromosomes, the additional criterion of the presence of a satellite is available (Table I), but in view of the apparent morphological variation of satellites, they and their connecting strands are excluded in computing the indices.

Table II shows the range of measurements determined by various workers.

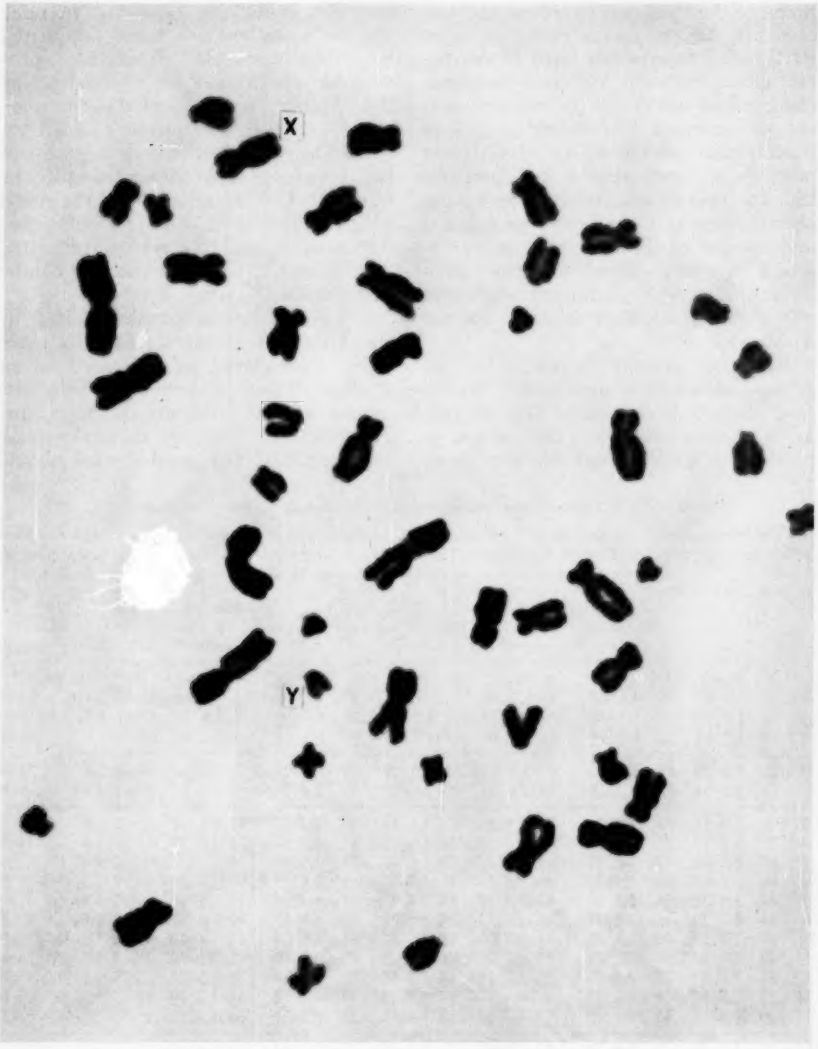
TABLE I. Conspectus of human mitotic chromosomes

Group 1-3	Large chromosomes with approximately median centromeres. The three chromosomes are readily distinguished from each other by size and centromere position.
Group 4-5	Large chromosomes with submedian centromeres. The two chromosomes are difficult to distinguish, but chromosome 4 is slightly longer.
Group 6-12	Medium sized chromosomes with submedian centromeres. The X chromosome resembles the longer chromosomes in this group, especially chromosome 6, from which it is difficult to distinguish. This large group is the one which presents major difficulty in identification of individual chromosomes.
Group 13-15	Medium sized chromosomes with nearly terminal centromeres ("acrocentric" chromosomes). Chromosome 13 has a prominent satellite on the short arm. Chromosome 14 has a small satellite on the short arm. No satellite has been detected on chromosome 15.
Group 16-18	Rather short chromosomes with approximately median (in chromosome 16) or submedian centromeres.
Group 19-20	Short chromosomes with approximately median centromeres.
Group 21-22	Very short, acrocentric chromosomes. Chromosome 21 has a satellite on its short arm. The Y chromosome is similar to these chromosomes.

CHROMOSOMES OF NORMAL HUMAN FEMALE

Figure 10

The X chromosome resembles the longer chromosomes in the medium-sized group numbered 6 to 12, especially chromosome 6, from which it is difficult to distinguish. (Photographs were originally published by J. H. Tjio and T. T. Puck, *Proceedings of the National Academy of Sciences*, 44:1229-1237, 1958.)



CHROMOSOMES OF NORMAL HUMAN MALE

Figure 11

The Y chromosome is very similar to the short, acrocentric chromosomes of the group numbered 21 to 22.

Some of the variation expresses the uncertainty due to measurement of relatively small objects, but many of the discrepancies between different workers' observations are due to the measurement of chromosomes at different stages of mitosis and to the effect of different methods of pretreatment and preparation for microscopic study. The ranges shown, therefore, represent the maxima and minima of the means found by different workers using different techniques. However, within any one worker's observations, the variations are not so broad.

Reference should be made to two other matters of nomenclature. In the first place, it is considered that no separate nomenclature for the groups is needed. It is considered that any group to which it may be necessary to refer will be a sequence of those designated by Arabic numerals. Hence, any chromosome group may be referred to by the Arabic numerals of the extreme chromosomes of the group, joined together by a hyphen, e.g., the group of the three longest chromosomes would be Group 1-3. This scheme has the merit of great flexibility. For instance, chromosomes X and 6 may be separated from Group 6-12 whenever they can be distinguished.

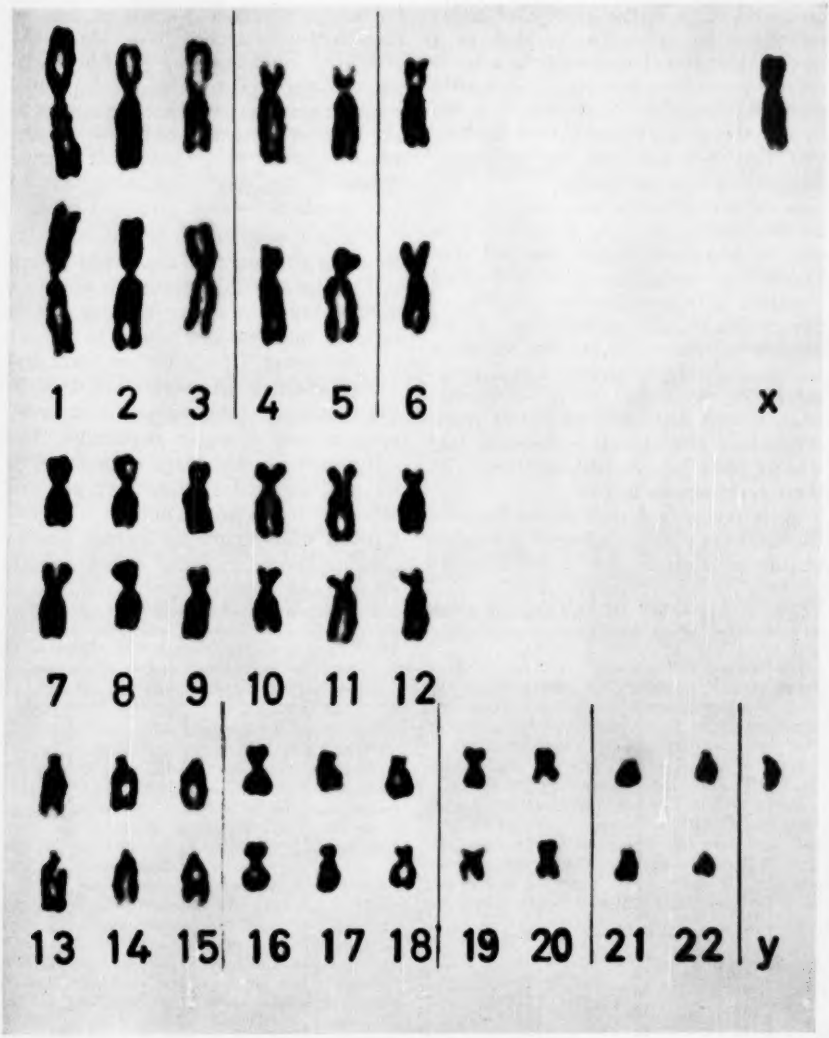
There is also the problem raised by the abnormal chromosomes which are being encountered in the more recent studies. Their nomenclature was discussed without a definite conclusion being reached. Broadly, it was agreed, however, that any symbol used should

TABLE II. Quantitative characteristics of the human mitotic chromosomes

All measurements were made from cells of normal individuals, except those made by Fraccaro and Lindsten, which included cases of Turner's Syndrome. The column A is the relative length of each chromosome, B is the arm ratio and C the centromere index, as defined in the text.

	Tjio and Puck[6]			Chu and Giles[2]			Levan and Hsu[5]			Fraccaro and Lindsten*			Lejeune and Turpin[4]*			Buckton, Jacobs and Harnden*			Range		
	A	B	C	A	B	C	A	B	C	A	B	C	A	B	C	A	B	C	A	B	C
1	90	1.1	48	90	1.1	48	85	1.1	49	82	1.1	48	87	1.1	48	83	1.1	48	82-90	1.1	48-49
2	82	1.6	39	83	1.5	40	79	1.6	38	77	1.5	40	84	1.5	40	79	1.6	38	77-84	1.5-1.6	38-40
3	70	1.2	45	72	1.2	46	69	1.2	45	65	1.2	45	67	1.2	46	63	1.2	46	63-72	1.2	45-46
4	64	2.9	26	63	2.9	26	63	2.7	27	62	2.6	28	62	2.6	28	60	2.6	28	60-64	2.6-2.9	25-28
5	58	3.2	24	58	3.2	24	59	2.6	28	60	2.4	29	57	2.4	30	57	2.4	30	57-60	2.4-3.2	24-30
X	59	1.9	34	57	2.8	26	52	1.6	38	54	1.6	38	58	2.2	32	51	1.7	37	51-59	1.6-2.8	32-38
6	55	1.7	37	56	1.8	36	56	1.7	37	54	1.6	38	56	1.7	37	56	1.6	38	54-56	1.6-1.8	36-38
7	47	1.3	43	52	1.9	35	51	1.9	35	50	1.7	37	51	1.8	36	50	1.7	37	47-52	1.3-1.9	35-43
8	44	1.5	40	46	1.7	37	48	1.6	39	47	1.7	37	48	2.4	29	46	1.5	40	44-48	1.5-2.4	29-40
9	44	1.9	34	46	2.4	29	47	1.8	36	45	2.0	33	47	1.9	35	44	2.1	32	44-47	1.8-2.4	32-40
10	43	2.4	29	45	2.3	30	45	2.0	33	45	2.6	28	45	2.6	27	44	1.9	35	43-45	1.9-2.6	27-35
11	43	2.8	26	44	2.1	32	44	2.2	31	43	2.2	31	44	1.6	39	43	1.5	40	43-44	1.5-2.8	31-40
12	42	3.1	24	43	3.1	24	42	1.7	37	43	1.7	37	42	2.8	27	42	2.1	32	42-43	1.7-3.1	24-37
13	35	8.0	11	32	9.7	10	32	5.0	16	34	4.8	17	33	6.8	14	36	4.9	17	32-36	4.8-9.7	10-17
14	32	7.3	12	34	9.5	9	37	4.0	20	35	4.4	19	32	7.0	13	34	4.3	19	32-37	4.3-9.5	9-19
15	29	10.5	9	31	11.9	8	35	4.7	17	33	4.6	18	31	10.0	9	34	3.8	22	29-35	3.8-11.9	8-22
16	32	1.8	36	27	1.6	38	30	1.4	42	31	1.4	42	29	1.4	41	33	1.4	41	27-33	1.4-1.8	31-42
17	29	2.8	26	30	2.1	33	29	2.4	30	30	1.9	35	29	3.1	23	30	1.8	36	29-30	1.8-3.1	23-36
18	24	3.8	21	25	3.8	22	25	2.6	28	27	2.5	29	26	4.2	19	27	2.4	29	24-27	2.4-4.2	21-29
19	22	1.4	41	22	1.9	34	24	1.2	45	25	1.3	43	22	1.4	42	26	1.2	45	22-26	1.2-1.9	34-45
20	21	1.3	44	19	1.3	44	21	1.2	45	23	1.3	43	20	1.2	45	25	1.2	46	19-25	1.2-1.3	40-46
21	18	3.7	21	15	6.8	13	13	2.5	28	19	2.5	29	15	2.3	31	20	2.5	29	13-20	2.3-6.8	13-31
22	17	3.3	23	12	6.0	14	16	2.0	33	17	2.3	30	13	4.0	20	18	2.7	27	12-18	2.0-6.0	14-33
Y	19	∞	0	11	∞	0	18	4.9	17	22	2.9	26	18	∞	0	18	4.9	17	11-22	2.9-∞	0-26

*Unpublished data.



KARYOTYPE OF NORMAL HUMAN MALE

Figure 12

Here the autosomes are serially numbered 1 to 22 as nearly as possible in descending order of length, as suggested by the Human Chromosome Study Group.

avoid incorporating a specific interpretation which was not reasonably established. It was suggested that arbitrary symbols, prefixed by a designation of the laboratory of origin, usually should be assigned to the abnormal chromosomes.

In this connection, two further requisites for coordination of research were

discussed. One is the storage of documentation for reference, perhaps in a central depository, additional to what it may be possible to publish. The other is the desirability that cultures be preserved by the satisfactory methods now used, so that they are available for reference, comparison and exchange.

Some consideration was also given to the desirability of using a uniform system for presenting karyotypes and idiograms, but recognizing that individual variation in taste is involved, rigidity of design was thought undesirable. However, it was recommended that the chromosomes should be arranged in numerical order, with the sex chromosomes near to but separated from the autosomes they resemble. It is desirable that similar ones be grouped together with their centromeres aligned.

It is recognized that choice between the different possible schemes of nomenclature is arbitrary, but that uniformity for ease of reference is essential. Hence, individual preferences have been subordinated to the common good in reaching this agreement. This human chromosome study group therefore agrees to use this notation and recommends that any who prefer to use any other scheme should, at the same time, also refer to the Standard System proposed here.

We are well aware of the wide interest in the work of this study group and realize that this meeting is merely a preliminary to a larger meeting. It is believed that two needs have to be met in this respect. One is for seminars and workshops at which workers in the field may exchange information; such seminars are best arranged regionally. The second need, which may come later, is for international conferences; and we believe that congresses and other organizations whose interests include human genetics should promote such meetings.

TABLE III. Synonymy of chromosomes as published by various workers

New chromosome number	Tjio and Puck[6]	Chu and Giles[2]	Levan and Hsu[5]	Ford, Jacobs and Lajtha[3]	Böök, Fraccaro and Lindsten[1]	Lejeune, Turpin and Gautier[4]
1	1	1	1	1	1	G1
2	2	2	2	2	2	G2
3	3	3	3	3	3	G3
4	4	4	4	4	4	G4
5	5	5	5	5	5	G5
6	6	6	6	6*	6	M1
7	7	7	7	(8)	7	M2
8	8	8	8	(9)	8	Md1
9	9	9	9	(11)	9	M3
10	10	10	10	10	10	Md2
11	11	11	11	(12)	11	M4
12	12	12	12	(13)	12	Md3
13	18	14	20	14	14	T1
14	19	15	18	15	15	T2
15	20	13	19	16	13	T3
16	13	17	15	19	16	C1
17	14	16	13	17	17	P1
18	15	18	14	18	18	P2
19	16	19	16	20	19	C2
20	17	20	17	21	20	C3
21	21	21	22	22	21	Vh
22	22	22	21	23	22	Vs
X	X	X	X	(7)	X	X
Y	Y	Y	Y	Y	Y	Y

*In the published idiogram the chromosomes of Group 6-12 (including X) were indicated by discontinuous lines and left unnumbered owing to the uncertainty of discrimination at that time. For the purpose of this table, these chromosomes have been assigned the numbers shown in brackets, in serial order of length.

Literature Cited

1. BÖÖK, J. A., M. FRACCARO and J. LINDSTEN. Cytogenetical observations in mongolism. *Acta Paediatrica* 48:453. 1959.

2. CHU, E. H. Y. and N. H. GILES. Human chromosome complements in normal somatic cells in culture. *Am. Jour. Human Genet.* 11: 63-79. 1959.

3. FORD, C. E., P. A. JACOBS and L. LAJTHA. Human somatic chromosome. *Nature* 181:1565. 1958.

4. LEJEUNE, J., R. TURPIN and M. GAUTIER. Le mongolisme, premier exemple d'aberration autosomique humaine. *Ann. Génét.* 2:41-49. 1959.

5. LEVAN, A. and T. C. HSU. The human idiogram. *Hereditas* 45:665-672. 1959.

6. TJIO, J. H. and T. T. PUCK. The somatic chromosomes of man. *Proc. Nat. Acad. Sci.* 44:1229-1237. 1958.

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AN INDEX OF CONSANGUINITY BASED ON THE USE OF THE SURNAME IN SPANISH-SPEAKING COUNTRIES

RICHARD F. SHAW*

THERE are several methods in genetics of deciding whether a given trait may be determined, either in whole or in part, by a recessive gene. One of these is based on the well-known fact that individuals homozygous for a rare recessive gene will very often prove to be children of parents who are related. In livestock this consanguinity can be measured by determining the average coefficient of inbreeding of the parents. In human populations in which close inbreeding is not the rule, the procedure is to determine what percentage of the parents are "related," or much better than that, what percentage are first cousins. I wish to propose another measure of relationship for man, an index of consanguinity based upon the Spanish custom of using both a maternal and a paternal surname.

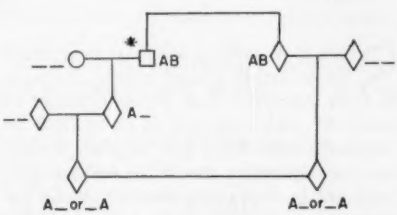
In Spanish-speaking countries each person has a pair of surnames. The first one is from the father and the second from the mother. Thus, individuals who under the English system would be called Juan Ortiz and Carmen Flores each have an additional name according to the Spanish system. The full names might be, for example, Juan Ortiz Rivera and Carmen Flores Figueroa. If Carmen marries Juan, all of their children will carry both the names Ortiz and Flores and in that order. A child of this family may decide to drop the name Flores for ordinary purposes, but he will know that he has two surnames and will give both if asked.

The names of cousins depend upon the sex of the two siblings which produced them, and since either sib has a 50 percent chance of being of either sex, the name combinations in a marriage of first cousins will be one of the following equally frequent types:

	Male × Female
Type 1	A_ × A_
2	A_ × _Ā
3	_Ā × A_
4	_A × _Ā

The important thing to notice is that the name "A" appears as one of the surnames of both individuals. In other words, *in every marriage of first cousins the spouses will have one surname alike.*

It will be apparent that not all couples having a surname in common will be first cousins, even though the converse is true. Many couples with names alike will be unrelated, but there is still considerable likelihood that they are related in some degree even if they are not first cousins. Consider this pedigree:



Here one extra individual occurs beyond those forming the chain of relationship in a first cousin marriage. If the individual with the

(Continued on page 230)

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DIPLOIDIZATION IN NICOTIANA TABACUM

A Study of the Yellow Burley Character

BILL J. STINES AND T. J. MANN*

SEGREGATING POPULATION

Figure 13

A normal green variety crossed with the burley variety Ky 16 produced both normal and burley plants. The stems of the burley plants are pale white and their leaves show more chlorosis than the leaves of normal plants.

SINCE *Nicotiana tabacum* originated as an amphidiploid hybrid, it might be expected that the inheritance of many of its characters is controlled by duplicate factors. If this be true, mutant recessive genes in one of the subgenomes may not be expressed phenotypically because of dominant genes in a duplicate locus of the other subgenome. Those characters which are governed by duplicate loci may become functionally diploidized through mutation — a common feature in amphidiploid species[2, 8].

Various workers have shown that a number of characters of tobacco have become functionally diploidized. Clausen

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and Cameron[4] found that the white seedling character was governed by duplicate recessive factors. While most of the varieties genetically analyzed were of the gametic genotype Ws_1Ws_2, some were found which differed from white seedling by a single factor. A phenotypically similar mutant (genetic homology has not been proven) was described by Gwynn[6]. In another case, Gwynn showed that duplicate recessive factors were responsible for the indeterminate growth habit, and that by crossing 402 and Bottom Special with the non-flue-cured variety, Culque Tinged, indeterminate plants could be recovered in the F_2 population. Mann and Weybrew[9] found that one of the two factors involved in alkaloid conversion in the amphidiploid *N. sylvestris* × *N. tomentosiformis* either lacked at the time of its synthesis, or has since lost, the ability to convert nicotine into nornicotine.

The fact that other characters have been shown to be governed by single-factor differences might suggest that diploidization has occurred. Several workers have described mutant characters in *N. tabacum* which gave monohybrid ratios in advanced generations[10-13].

The demonstration by Henika[7] that the white burley character (called yellow burley character by Clausen and Cameron[3]) was best explained on the basis of duplicate recessive factors has aroused curiosity as to the genetic constitution of nonburley varieties of *N. tabacum* with respect to this character. The objective of this study was to determine the genotypes (with respect to the above character) of 20 nonburley varieties, selected to represent flue-cured, Maryland, and Turkish types.

Dr. H. E. Heggestad (personal communication) has found that the Maryland variety, Catterton, in hybrid combination with a burley variety segregated in a ratio of 3 green : 1 burley in the F_2 population. This indicates that Catterton has been reduced to the duplex condition. However, it is possible that the recessive locus of Catterton was due to hybridization with burley and not mutation[1].

Materials and Methods

Monomeric and dimeric classification

In 1957 20 normal green varieties (given in the results) were crossed with the burley variety Ky 16 (yb_1yb_1 yb_2yb_2) to determine whether these normal green varieties differed from burley by duplicate factors or by a single factor.

Four replications of the F_2 and testcross seed were sown in eight-inch pots (approximately 100 seed per pot) filled with a sterilized mixture of three parts sand to one part vermiculite. The seedlings were fertilized with Hoagland's solution (solution 1) twice weekly for approximately three weeks. The solution was then discontinued, and the plants were grown in a semistarved condition which induced the burley character to become visible much earlier than under normal conditions. Classification was based on the color of the stems and the yellowing of the leaves. The stems of the burley plants were pale white in comparison with the green stems of other plants, and the leaves of the burley plants showed more chlorosis than did the other plants (Figure 13).

In advanced generations, those varieties of the gametic genotype Yb_1Yb_2

TABLE I. Diagnostic behavior of varieties of *N. tabacum*, when crossed with two tester lines

Tester	Constitution of unknown		
	Yb_1Yb_2	Yb_1yb_2	yb_1Yb_2
G1 (Yb_1yb_2)	F_2 all green	F_2 all green	F_2 15:1
	TC* all green	TC all green	TC 3:1
G2 (yb_1Yb_2)	F_2 all green	F_2 15:1	F_2 all green
	TC all green	TC 3:1	TC all green

*TC—Testcross of F_1 hybrid × Ky 16.

would be expected to segregate in a 15 green : 1 burley ratio in the F_2 and a 3 green : 1 burley ratio in the testcross to Ky 16. Those varieties of the gametic genotype Yb_1yb_2 or yb_1Yb_2 would be expected to segregate in a 3 green : 1 burley ratio in the F_2 and a 1 green : 1 burley ratio in the testcross.

Location of factors yb_1 and yb_2 using tester lines

One cannot distinguish between the gametic genotypes Yb_1yb_2 and yb_1Yb_2 in advanced generations of varietal hybrids with Ky 16. The distinction can be made by crossing with two tester lines—G1(Yb_1yb_2) and G2(yb_1Yb_2). The expected results are given in Table I.

Crosses involving the tester lines were made on plants other than those crossed with Ky 16, and four F_1 plants were grown to maturity. In most cases only one F_1 plant was selected to be selfed and testcrossed, but in a few cases two plants were selected because of a lack of flowers on one of the plants. Testcross and F_2 seed were sown, and the seedlings were classified about eight weeks later.

Monosomic test and intercrosses

To verify the results found using the tester lines, selected plants of each variety were also crossed with a monosomic B plant and a monosomic O plant. These monosomics were used since it has been established by Clausen and Cameron[3] that the factor yb_1 is located in chromosome B and yb_2 in chromosome O. Only the F_1 seed from varieties which differed from burley by a single factor were utilized.

Forty-four plants of each F_1 hybrid were planted in the field and two F_1 haplo B plants and two F_1 haplo O plants of each monosomic hybrid were selected. The selection was based first on morphological characteristics and later verified cytologically. The expected results of the monosomic test are given in Table II.

The testcross seed were sown in the greenhouse, and the seedlings were classified as to normal green versus burley about eight weeks later.

Intercrossing the varieties might be expected to provide supplementary information concerning the genotype. Had diploidization occurred and different subgenomes been involved, burley segregants would be expected in a 15 green : 1 burley ratio in the F_2 and 3 green : 1 burley ratio in the testcross. Most of the possible one-way intercrosses were made, and these were handled much the same way as the other crosses.

Results and Discussion

Monomeric and dimeric classification

It is shown in Table III that 18 of the 20 varieties in hybrid combination with Ky 16 segregated in an approximate 15 green : 1 burley ratio in the F_2 and approximately 3 green : 1 burley in the testcross. The dimeric segregation ratios indicate that these varieties are dominant at both loci.

The sum of chi-squares is highly significant for the testcross generation, which indicates a deviation from the expected ratio with no distinction between excess and deficiency. The sum of chi-squares is nonsignificant for the F_2 generation.

The pooled chi-square is nonsignificant for the F_2 population, but for the testcross it is highly significant, indicating a predominance of deviations with a common sign (in this case a deficiency of recessives). Throughout this study

TABLE II. Diagnostic behavior of varieties of *N. tabacum*, when crossed with two monosomics

	Constitution of unknown		
	Yb_1Yb_2	Yb_1yb_2	yb_1Yb_2
Monosomic B	TC* all green	TC some burley†	TC all green
Monosomic O	TC all green	TC all green	TC some burley†

*TC—Testcross of selected F_1 haplo plant with Ky 16.

†Burley character expressed in approximately half of the monosomic plants of the testcross.

there tended to be a deficiency of recessives, which might be expected since Henika[7] has demonstrated a differential zygotic viability between dominant green and recessive yellow burley genotypes.

The signs of the deviations are recognized and their inconsistency evaluated by the heterogeneity chi-square. The deficiency of recessives tended to be constant with all varieties, as shown by the

TABLE III. Dimeric classification. F_2 and testcross populations tested for goodness of fit to a 15:1 ratio in the F_2 and a 3:1 ratio in the testcross

Population	Green	Burley	χ^2
(Cabbage × Ky 16) x	306	12	3.328
(Cabbage × Ky 16) × Ky 16	357	97	3.198
(Bottom Special × Ky 16) x	377	24	0.048
(Bottom Special × Ky 16) × Ky 16	323	85	3.778
(402 × Ky 16) x	390	23	0.327
(402 × Ky 16) × Ky 16	300	85	1.753
(Coker 139 × Ky 16) x	421	23	0.867
(Coker 139 × Ky 16) × Ky 16	224	67	0.606
(Coker 140 × Ky 16) x	315	16	1.133
(Coker 140 × Ky 16) × Ky 16	276	78	1.661
(400 × Ky 16) x	508	32	0.097
(400 × Ky 16) × Ky 16	358	126	0.275
(Big Jim × Ky 16) x	336	18	0.820
(Big Jim × Ky 16) × Ky 16	362	112	0.475
(Cash × Ky 16) x	435	24	0.817
(Cash × Ky 16) × Ky 16	442	124	2.886
(Mammoth Gold × Ky 16) x	569	34	0.385
(Mammoth Gold × Ky 16) × Ky 16	315	70	9.545**
(Yellow Special A × Ky 16) x	542	40	0.385
(Yellow Special A × Ky 16) × Ky 16	342	92	3.346
(Dixie Bright 101 × Ky 16) x	577	36	0.149
(Dixie Bright 101 × Ky 16) × Ky 16	450	104	11.458**
(Exp. Line 3 × Ky 16) x	516	40	0.846
(Exp. Line 3 × Ky 16) × Ky 16	402	105	4.976*
(Turkish Smyrna 23 × Ky 16) x	494	32	0.025
(Turkish Smyrna 23 × Ky 16) × Ky 16	282	83	0.995
(Turkish Samsoun 15 × Ky 16) x	220	12	0.460
(Turkish Samsoun 15 × Ky 16) × Ky 16	298	84	1.846
(Ravnjak × Ky 16) x	369	21	0.498
(Ravnjak × Ky 16) × Ky 16	409	112	3.409
(New Guinea × Ky 16) x	618	40	0.033
(New Guinea × Ky 16) × Ky 16	535	134	8.814**
(White Peru × Ky 16) x	678	45	0.001
(White Peru × Ky 16) × Ky 16	394	113	1.989
(Robinson Med. Broad. × Ky 16) x	284	17	0.186
(Robinson Med. Broad. × Ky 16) × Ky 16	318	88	2.394
Sum of chi-squares (15:1 ratio)			10.405
Sum of chi-squares (3:1 ratio)			63.404**
Pooled chi-square (15:1 ratio)	7955	489	3.035
Pooled chi-square (3:1 ratio)	6387	1759	50.417**
Heterogeneity (15:1 ratio)			7.370
Heterogeneity (3:1 ratio)			12.987

*Significant at 5 percent level.
**Significant at 1 percent level.

test of heterogeneity being nonsignificant for both F_2 and testcross material.

The four varietal hybrids which deviated from the expected ratio in the testcross were reseeded. In the second seeding, the results fitted the expected 3 green : 1 burley ratio except for the New Guinea hybrid. Again the deviation was due to a deficiency of recessives. It is probable that many of the recessive genotype seed failed to germinate.

Two Maryland varieties, Posey and Catterton, in hybrid combination with Ky 16 segregated in an approximate 3 green : 1 burley ratio in the F_2 and approximately 1 green : 1 burley in the testcross with Ky 16 (Table IV). The monomeric segregation ratios indicate that Posey and Catterton have diploidized for the yellow burley character. This is in agreement with the results observed by Heggestad (personal communication) who studied the F_2 segregation ratio of Catterton crossed with a burley variety.

It was thought advisable to check the tester lines to determine whether they gave the expected ratios. Testcross and F_2 results given in Table IV show a good fit to the expected 3:1 and 1:1 ratios.

The fact that two of the three Maryland varieties included in this study differed from burley by a single factor might lend support to the statement by Garner *et al.*[5] that burley arose as a mutation from the Maryland type. However, this does not preclude the possibility that diploidization of these varieties might be a result of hybridization of Maryland and burley types and not mutation[1].

TABLE IV. Monomeric classification. F_2 and testcross populations tested for goodness of fit to a 3:1 ratio in the F_2 and a 1:1 ratio in the testcross

Population	Green	Burley	χ^2
(Posey × Ky 16) x	457	145	0.268
(Posey × Ky 16) × Ky 16	427	384	2.280
(Catterton × Ky 16) x	495	152	0.784
(Catterton × Ky 16) × Ky 16	417	394	0.652
(G1 × Ky 16) x	75	16	2.670
(G1 × Ky 16) × Ky 16	66	59	0.392
(G2 × Ky 16) x	105	36	0.021
(G2 × Ky 16) × Ky 16	65	55	0.833

Location of factors yb_1 and yb_2 using tester lines

When those varieties which gave dimeric segregation ratios in hybrid combination with Ky 16 were crossed with the two tester lines, no burley segregants were observed in advanced generations except in the case of Bottom Special. The unexpected segregation in the F_2 and testcross generations (124:1 in the F_2 and 98:12 in the testcross) of the Bottom Special hybrid with the tester G1 (Yb_1yb_2) can be explained by assuming that the Bottom Special parent used in this series of crosses was heterozygous for one of the factors and homozygous dominant for the other (Yb_1yb_1 Yb_2Yb_2). On the basis of this proposed genotype, two F_1 genotypes would be expected, Yb_1Yb_1 Yb_2yb_2 and Yb_1yb_1 Yb_2yb_2. While no segregation would be expected in advanced generations involving the genotype Yb_1Yb_1 Yb_2yb_2, dihybrid ratios would be expected with the other genotype.

On the assumption that the F_1 plants were identical genetically and lacking sufficient flowers on one plant, two F_1 plants were used in the production of F_2 and testcross seed. The F_2 seed from the two plants was bulked as was the testcross seed. The bulked seed was later sown and the resulting seedlings were classified. The two F_1 parent plants selected must have been of different genotypes. Since the seed from the two parent plants was not kept separate, nondiscernible ratios resulted.

A loss of a chromosome would not explain the observed results. If the Bottom Special plant used had lost a chromosome or was a

TABLE V. Results from crosses with two tester lines tested for goodness of fit to indicated ratios

Population	Green	Burley	χ^2
A. Expected 15:1 ratio in the F_2 and 3:1 ratio in the testcross.			
(Posey × G2) x	194	8	1.807
(Posey × G2) × Ky 16	195	64	0.012
(Catterton × G2) x	335	20	0.230
(Catterton × G2) × Ky 16	313	95	0.641
(G1 × G2) x	134	7	0.398
(G1 × G2) × Ky 16	119	39	0.008
B. Expected all green in the F_2 and the testcross.			
(Posey × G1) x	365	0	
(Posey × G1) × Ky 16	381	0	
(Catterton × G1) x	486	0	
(Catterton × G1) × Ky 16	565	0	
G1 x	172	0	
G2 x	172	0	

monosomic and was in turn crossed with the tester, G1 (Yb_1yb_2), normal and monosomic plants might have been obtained. The normal plants in this population would not segregate for the burley character in advanced generations. Monosomic plants in hybrid combination with the above tester would have segregated for the burley character in the testcross to Ky 16, but no burley segregants would have been possible in the F_2 since burley segregants in the F_2 would have had to be nullisomic. Viable nullisomics in *N. tabacum* have not been reported so far as is known by the writers.

In a second seeding of the Bottom Special hybrids, similar results were observed.

The assumption that the Bottom Special plant used was heterozygous at one locus is not unreasonable even though tobacco is a self-pollinated crop. The finding of a heterozygote at one locus would be expected if diploidization through mutation was in progress. There is also the possibility of outcrossing in tobacco. Gwynn[6] obtained evidence that Bottom Special was heterozygous for one of the factors concerned with chlorophyll production.

When Posey and Catterton were crossed with the tester, G2 (yb_1Yb_2), a segregation ratio of approximately 3 green : 1 burley was observed in the testcross and approximately 15 green : 1 burley in the F_2. In hybrid combination with the other tester, all of the plants in advanced generations were green. The results recorded in Table V indicate that Posey and Catterton are of the genotype Yb_1Yb_1 yb_2yb_2.

The results of intercrossing and selfing the tester lines indicate that these lines are homozygous and differ from burley by a single factor but in different subgenomes (A_1a_2 as contrasted to a_1A_2).

It is of interest to note that the locus apparently undergoing diploidization in Bottom Special was not the one involved in Posey and Catterton. Posey and Catterton were recessive at the *yb* locus in chromosome O while in Bottom Special heterozygosity of the *yb* locus in chromosome B was postulated.

Monosomic test and intercrosses

Further confirmation of the genotypes of Posey and Catterton was obtained by crossing these varieties with monosomics B and O. The results obtained were in agreement with the results when using the tester lines.

Since none of the intercrosses segregated for the burley character in advanced generations, it was concluded that Posey and Catterton must be of the same genotype with respect to the character under study. This, too, is in agreement with the results of the other tests.

Summary and Conclusions

The inheritance of the chlorophyll-deficient yellow burley character in crosses of the burley variety Ky 16 with normal green tobacco was studied. The results from advanced generations indicated that 18 of the 20 varieties tested still possessed the unmodified duplicate dominant condition. Two Maryland varieties, Posey and Catterton, gave monomeric segregation ratios in the F_2 and testcross generations. Monosomic analysis and use of two tester lines demonstrated that both of these varieties were recessive at the *yb* locus in chromosome O.

Bottom Special when crossed with one of the tester lines gave unexpected ratios in both the F_2 and testcross generations. It was hypothesized that the Bottom Special parent plant used was heterozygous at the *yb* locus in chromosome B, suggesting diploidization in process as a result of mutation from *Yb* to *yb* at this locus.

Literature Cited

1. Bowling, J. D., J. E. McMurtrey and D. E. Brown. Variety, description, and performance tests with Maryland tobacco. *University of Maryland Bul.* A73. 1952.

2. Clausen, R. E. Polyploidy in *Nicotiana*. *Amer. Nat.* 75:291-306. 1941.

3. ———— and D. R. Cameron. Inheritance in *Nicotiana tabacum*. XVIII. Monosomic analysis. *Genetics* 29:447-477. 1944.

4. ———— and ————. Inheritance in *Nicotiana tabacum*. XXIII. Duplicate factors for chlorophyll production. *Genetics* 35: 4-10. 1950.

5. Garner, W. W., H. A. Allard and E. E. Clayton. Superior germ plasm in tobacco. *U.S.D.A. Yearbook of Agriculture*. pp. 785-830. 1936.

6. Gwynn, G. R. Mode of inheritance of certain quantitative and qualitative characters in *Nicotiana tabacum*. Unpublished M.S. Thesis. North Carolina State College. Raleigh. 1954.

7. Henika, F. S. The inheritance of the white burley character in tobacco. *Jour. Agri. Res.* 44:477-493. 1932.

8. Hutchinson, J. B. On the occurrence and significance of deleterious genes in cotton. *Jour. Genet.* 47:272-289. 1946.

9. Mann, T. J. and J. A. Weybrew. Inheritance of alkaloids in hybrids between fluecured tobacco and related amphidiploids. *Tobacco* 146(12): 20-25. (*Tobacco Sci.* 2:29-34). 1958.

10. Nolla, J. A. B. Inheritance in *Nicotiana*. I. Study of the glaucous and the yellow characters in *N. tabacum*. *Jour. Agri. Univ. Puerto Rico* 18:443-462. 1934.

11. Valleau, W. D. Yellow Crittenden—a mutant in dark tobacco. *Tobacco* 144(25): 20-21. (*Tobacco Sci.* 1:91-92). 1957.

12. ————. Genetic veinbanding—a white-flowered tobacco mutant. *Tobacco* 146, (10): 19-22. (*Tobacco Sci.* 2:20-22). 1958.

13. ————and G. W. Stokes. Virescent—a chlorophyll deficiency in tobacco. *Tobacco* 145(20):20-21. (*Tobacco Sci.* 1:175-176). 1957.

MAHOGANY, A RECESSIVE COLOR MUTATION IN LINKAGE GROUP V OF THE MOUSE

PRISCILLA W. LANE AND MARGARET C. GREEN*

SINCE the early part of the century various investigators have studied the inheritance of factors which darken the coat color of mice. The first studies were conducted on darkly pigmented yellow mice. In 1916 Little[6] attempted to modify the agouti pattern in mice by crossing the darkest heterozygous agouti animals selected from a "sooty yellow" stock to nonagouti animals from the same stock. Both the agouti and nonagouti animals grew darker as the selected crossing continued. Dunn[3] in 1920 outcrossed a dark yellow stock to an agouti stock and was able by selection to increase the darkness of the agouti offspring in subsequent generations.

In 1934 Barrows[1] studied dorsally darkened wild-type mice not derived from any modified yellow stock. His results suggested that one or more genetic factors were responsible for the darkened phenotype and that one of the modifying factors was loosely linked with nonagouti. Mather and North[7] in 1940 reported a semidominant gene, umbrous, which segregated independently of agouti. This was the first of the dark phenotypes to show single-factor Mendelian inheritance. Barrows and Mather and North found that homozygous nonagouti animals were unaffected by the presence of the darkening factor. Recently Robinson[9] was able to isolate a gene of the umbrous type which was transmitted as a simple dominant. This gene also segregated independently of agouti.

Several years ago mice of unusually dark wild-type color appeared in this laboratory from parents of unknown origin. These animals resembled the descriptions of umbrous mice given by Barrows and by Mather and North. The central dorsal hairs were considerably darker than those of normal agouti mice, the yellow ticking characteristic of agouti being considerably reduced. The darkening was present but less intense in the lateral hairs. The ventral hairs were almost solid grey with no yellow ticking. In addition, the ears and tail appeared more deeply pigmented than those of normal wild-type mice. This paper presents evidence that the observed phenotype is due to a recessive gene (mahogany *mg*) located in the fifth linkage group.

Results

The first pair of mahogany mice were mated together, and all offspring resembled their dark parents. This suggested a new recessive color mutation rather than a repetition of the dominant umbrous mutation previously reported. The mahogany mice were bred brother by sister for almost 20 generations and showed uniformly the dark agouti phenotype. Inbred mahogany males were then outcrossed to C57BL/6J (*aa*) females, and all the F_1 offspring were of wild-type coat color. When these F_1 mice were mated *inter se*, three classes of animals rather than the expected four classes were noted. It was apparent that either the nonagouti and the nonagouti mahogany animals were indistinguishable or the nonagouti mahogany class was missing altogether. In addition, inexperience at that time in dis-

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tinguishing mahogany agouti from agouti led to the classification of some mahogany agoutis as agouti.

In order to determine whether the nonagouti mahogany phenotype was indistinguishable from normal nonagouti and whether mahogany was segregating as expected in the presence of nonagouti, 20 F_2 nonagouti females were mated to inbred mahogany males. Fifteen of these females produced no mahogany offspring while five produced half mahogany and half nonmahogany offspring. If the mahogany phenotype were due to a recessive gene segregating independently of the agouti locus, one-fourth of the females should have produced no mahogany offspring, one-half should have produced mahogany and nonmahogany, and one-fourth should have produced only mahogany. The deviation from expectation suggested linkage of agouti and mahogany.

A pair of mahogany offspring from one of the nonagouti females which produced half mahogany were mated together to produce homozygous nonagouti mahogany animals. A male of this type was then mated to F_1 females of the original cross to C57BL/6J. These matings produced 2 normal agoutis, 24 mahogany agoutis, and 34 nonagoutis. This outcome confirmed the previous indication of linkage between agouti and mahogany.

A closer examination of the nonagouti offspring of crosses of this type revealed that it was possible to distinguish mahogany from nonmahogany. The coat of the mahogany mice was coal black and no white hairs were evident behind the ears or around the perineum as are evident in C57BL/6 mice. The tail, feet, and ears were blacker than in normal nonagouti animals. In general appearance nonagouti mahogany mice resemble the nonagouti mice selected for dark modifiers by Little[6], and the mice homozygous for extreme nonagouti described by Hollander and Gowen[5].

In order to measure more exactly the linkage between agouti and mahogany and to determine the position of mahogany in linkage group V further crosses were performed. The results are given in Table I. Mahogany segregates normally in all crosses except one. For all crosses combined there is no significant deviation from expectation ($\chi^2 = 0.53$, df = 1, P = .50-.30), and since the heterogeneity between crosses is not significant ($\chi^2 = 13.16$, df = 7, P = .10-.05), we may regard the abnormal segregation as due to chance.

The three-point cross with ragged (*Ra*) shows the order of the three genes to be *Ra-a-mg*. This puts *mg* very close to wellhaarig (*we*), pallid (*pa*), and rough (*ro*). The recombination between *Ra* and *a* estimated from the totals is 20.6 ± 1.4 percent, which agrees reasonably well with previous estimates[2, 8]. The recombination between *a* and *mg* is 9.8 ± 1.2 percent in males and 13.2 ± 1.0 percent in females. The difference between the sexes is significant ($\chi^2 = 4.51$, df = 1, P = .05-.02) and is in the same direction as that observed by

TABLE I. Results of linkage crosses with mahogany, *mg*

Mating ♀	♂	Offspring ++	+mg	a+	amg	Total	% recombination a-mg	Ra-a	χ^{2mg*}
amg/amg	a+/+mg	7	80	80	10	177	9.6±2.2		0.05
a+/+mg	amg/amg	37	235	236	41	549	14.2±1.5		0.02
amg/amg	++/amg	59	6	7	45	117	11.1±2.9		1.92
++/amg	amg/amg	54	5	8	38	105	12.4±3.2		3.44
		Raa+	a+ +mg	a+ ++	Ra++mg	++ ++	Raamg	+amg	Ra++ ++	Total			
+amg/+amg	Raa+/++mg	73	70	24	25	10	9	2	0	213	9.9±2.0	23.9±2.9	0.00
Raa+/++mg	+amg/+amg	105	79	28	24	18	14	0	1	269	12.3±2.0	19.7±2.4	4.55
+amg/+amg	Ra++/+amg	0	0	5	9	9	18	61	52	154	9.1±2.3	17.5±3.1	3.14
Ra++/+amg	+amg/+amg	1	1	15	9	16	26	76	69	213	12.2±2.2	20.7±2.8	0.57

*χ^2 for segregation of *mg*.

Fisher and Landauer[4] for this region of linkage group V.

Summary

This paper describes a new recessive gene of the umbrous type called mahogany, *mg*, located in linkage group V about 12 units from agouti. Recombination with *a* is 9.8 ± 1.2 percent in males and 13.2 ± 1.0 percent in females. The linear order of the two loci and *Ra* and the approximate recombination percentages are as follows:

Ra—21—*a*—12—*mg*

Literature Cited

1. Barrows, E. F. Modification of the dominance of agouti to nonagouti in the mouse. *Jour. Genetics* 29:9-15. 1934.
2. Carter, T. C. and R. J. S. Phillips. Ragged, a semidominant coat texture mutant in the house mouse. *Jour Hered.* 45:151-154. 1954.
3. Dunn, L. C. The sable varieties of mice. *Amer. Nat.* 54:247-261. 1920.
4. Fisher, R. A. and W. Landauer. Sex differences of crossing-over in close linkage. *Amer. Nat.* 87:116. 1953.
5. Hollander, W. F. and J. W. Gowen. An extreme nonagouti mutant in the mouse. *Jour. Hered.* 47:221-224. 1956.
6. Little, C. C. The occurrence of three recognized color mutations in mice. *Amer. Nat.* 50:335-349. 1916.
7. Mather, K. and S. B. North. Umbrous: a case of dominance modification in mice. *Jour. Genetics* 40:229-241. 1940.
8. Parsons, P. A. Additional three-point data for linkage group V of the mouse. *Heredity* 12:357-362. 1958.
9. Robinson, R. Sable and umbrous mice. *Genetica* 29:243-255. 1959.

An Index of Consanguinity

(*Continued from page 221*)

asterisk is a male, as shown, the paternal last name is inherited exactly as before, and the spouses will have a name alike. That individual will actually be a male in half the pedigrees of this kind. Thus, marriages of first cousins once removed have a 50 percent chance of being detected and included in the proposed index. In the same way, the probability of detection is reduced by one-half for each additional ancestor who appears in the pedigree.

The method, clearly, is a statistical one. Although the names will not tell whether a given marriage is consanguineous, when large numbers of marriages are considered those in which a surname is the same for the two spouses will be consanguineous much more often than the others. The index, or measure, of consanguinity proposed here is thus simply the percentage of marriages in which the parents have one or more surnames alike.

It is true that the surname index presents some difficulties not encountered in the method of determining first cousin marriages by interview or through genealogies. If very common surnames are more abundant in one region than another, the surname index would vary regionally irrespective of any variation in consanguinity. Also, an exact formula for testing the hypothesis of inheritance by a single autosomal recessive is available for studies made on frequencies of first cousins, while such a formula based on the index under discussion is not available.

These difficulties probably would not be too hard to resolve, but even without refinements the proposed index can be used to determine whether individuals with a given trait tend to be from consanguineous parents. It is only necessary to compare the index for affected individuals with that of the population in which they occur or of an appropriate control group. A positive finding in such a study may be taken as evidence of recessive inheritance of one form or another.

The great advantage of using the index based on name combinations is that in many countries vast amounts of the required data are automatically collected or could be with little extra trouble. Where data of this kind are already available they should be used to investigate diseases of uncertain etiology, since evidence of recessive inheritance would be an important step in their genetic analysis. In any hospital where an accurate diagnosis is available, it is only necessary to ask patients for the full names of their parents in order to have the basic data for this kind of investigation.

MULTIPLE LIPOMATOSIS IN DAIRY CATTLE

J. L. ALBRIGHT*

INHERITED abnormal characteristics that appear in later life have to date been more common in humans than in domestic animals. Such conditions as progressive spinal muscular dystrophy, diabetes, exoderma pigmentosum, glioma retinae, and infantile amaurotic idiocy have received considerable attention due to their severity, appearance, and the time in the life cycle in which they occur[2].

A review of the literature on genetic abnormalities and lethals in dairy cattle reveals two conditions that develop with age and physiological changes. The condition of strabismus (cross-eyes) in Jersey cattle is not evident at birth but is identifiable by 12 months of age[11]. The simple recessive trait of digital anomaly follows a similar pattern[9]. Calves manifesting this abnormality become lame when two to four months of age, and after onset, the condition is progressive until, at 18 months of age, standing or walking causes obvious discomfort and the animals "walk" on their knees.

Among other species of farm animals, swine are prone to inherited abnormal developmental characteristics. For example, hemophilia has been observed in this species[2]. The condition is not apparent at birth, and its full expression is not witnessed until the pig is three or four months of age. Another inherited anomaly in swine is excessive fatness, whereby pigs become excessively fat at 70 to 150 pounds and die[2]. Of lesser economic consequence in swine are the conditions of kinky tails and melanotic tumors. Kinky tails are evident at birth and become more rigid with development. Melanotic tumors are small at birth and enlarge with age.

This paper reports an unusual characteristic of delayed development recently observed in two Holstein-Friesian dairy herds.

Description and Morphology

Large "growths" in the perineal area of full sibs are pictured in Figure 14. These growths were not observed until the animals were approximately three and one-half years of age, but from that age the growth in each became progressively larger. The male and female in Figure 14 were five and seven years old, respectively, when photographed.

The growths measured approximately nine inches horizontally, eight inches vertically, and two inches in depth. Apparently, they are attached to the musculature of the right leg. Autopsy would be required to establish the actual mode of attachment.

Tissue sections of the growths were obtained on the day the pictures were taken. The specimens were immediately frozen following surgical removal, and microtome preparations were made and examined microscopically. The growths in each case were diagnosed as adipose tissue and further classified as lipomas of the subcutaneous tissue.

The tissue sections showed strands of collagen from the dermis mixed with occasional fibrocytes containing a few endothelial-lined capillary spaces and large lobulated masses of atypical fat. The fat cells were of the adult type but showed almost complete lack of support-

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ing fibrous septa. There was no appearance of malignant degeneration and no sign of inflammation.

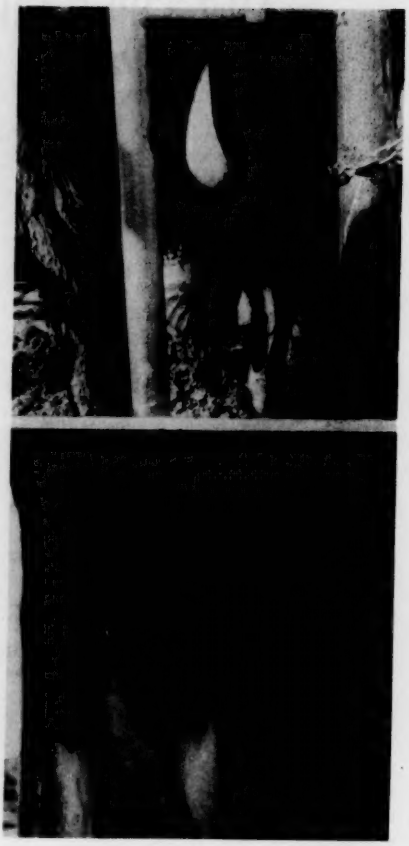
Both of these animals exhibited evidence of lipomatosis in that they possessed fat-like deposits behind their right front legs in the lower region of the chest area. These "growths" were smaller and not as apparent as the growths pictured in Figure 14. The most conspicuous was approximately three inches across and four inches in length. Each of the full sibs had one other smaller growth in the same region.

The right and left sides of the cow's mammary system are illustrated in Figure 15. It should be noted that the right half of the udder is considerably deeper than the left. The right side has deepened in relation to the growth of the aforementioned lipoma (Figure 14). This unusual depth in the right front quarter is apparent regardless of stage of lactation. Because of the unusually large size of the quarter during the dry period, palpation was performed to detect if mastitis, scar tissue, or some other udder abnormality was present, but none was detected. Tissue sections of the fore udder were not obtained, but it is probable that part of the observed imbalance was due to an accumulation of adipose tissue. The imbalance progressed during lactation.

Figure 16*A-C* shows the cow at three, five, and seven years. The lack of udder lipomatosis at three years and the progressive nature of the condition as the cow grows older is apparent.

The full sibs exhibiting multiple lipomatosis were from a high-producing family. The cow and her dam ranked in the upper one percent of the breed in terms of butterfat production with yearly records of more than 1000 pounds. The bull in Figure 14 had no daughters in milk.

The full sibs and their dam had a case history of low fertility. Because of the bull's continued low fertility and high number of services per conception, semen quality tests were initiated prior to the biopsy technique. During a consecutive four-week collection period, volume,



PERINEAL LIPOMA

Figure 14

These two Holstein-Friesian full sibs, illustrating lipoma in the perineal region, were approximately three and one-half years old when the lipomas were first discovered. From that age the growth in each became progressively larger.

motility, and concentration of semen were all within the normal range[6]. During the same period, morphological tests were conducted and abnormal sperm ranged from 21.3 to 44 percent, with an average of 35.3 percent. One might assume that the lipoma which is adjacent to the testes had some influence on fertility. However, upon palpation, this sire exhibited certain hypoplastic tendencies,

MAMMARY GLAND LIPOMATA

Figure 15

The right and left sides of the afflicted cow's mammary gland show the extent of udder lipomata (also pictured in Figures 14 and 16).

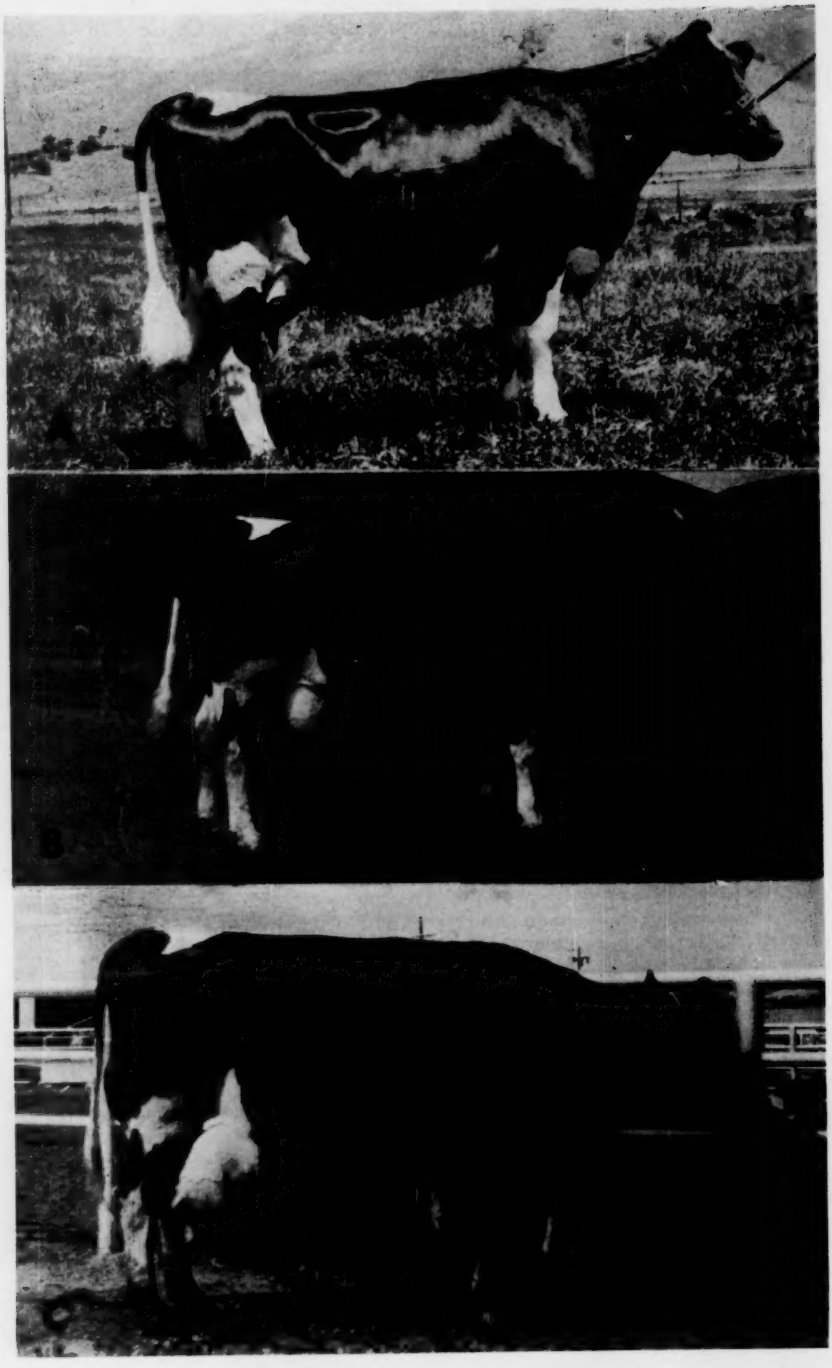
i.e., smaller left testis and shrunken scrotum. Hypoplasia, a recessive autosomal gene with incomplete penetrance, reduces directly or indirectly the fertility of the animal from the normal condition[1, 3].

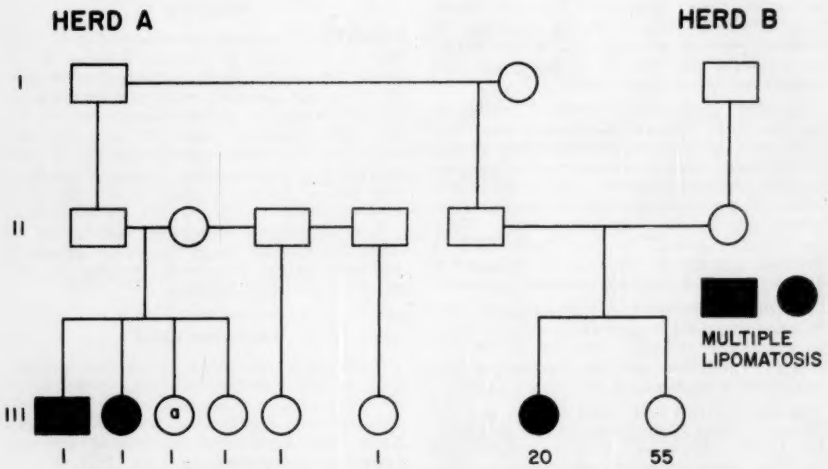
A second herd exhibited similar tendencies of unusual depth of udder and multiple lipomatosis. According to the owner, approximately 20 of his 75 cows possessed unusually large fat deposits in the rib areas of their bodies. In the case of one cow, fatty deposits were conspicuous in her mammary system as she approached maturity. Further observation revealed that her udder showed more imbalance and fat deposition than the cow in Figure 15. Both of these cows with large fat deposits in their mammary systems were outstanding milk and butterfat producers before the advancement of their affliction. Slaughter and autopsy of two cows in this group of 20 indicated that the deposits were primarily adipose tissue distributed throughout the internal and external regions of the body. Subsequent microscopic evaluations confirmed the early diagnosis. This group of cows were daughters of a bull which had the same paternal grandsire as the aforementioned full sibs (Figure 14).

Isolated cases of lipomata in humans are found frequently in the general population, but hereditary multiple lipomata are relatively rare[5, 8, 12, 13]. In a recently reported kindred, lipomata first appeared at around 35 years of age[13]. The age of onset varied somewhat, with one case being reported at nine years. Lipomata are normally painless, although one individual reported pain when the tumors first appeared. In some cases lipomata first appeared in regions of the body which regularly came in contact with some object such as a desk. The tumors continued to grow (unless removed) until they became as large as a hen's egg. In no instance within the kindred did any of the lipomas become malignant.

In animals, it is interesting to note that Johansson has observed lipomatosis in an inbred strain of rabbits in Sweden[7]. The fat-like tumors were found at random throughout their bodies. Heredity-linked tumors in mice[10] and Drosophila[4] have been observed; however, their specific tissue composition was not checked and it is not known if they are lipomata or should be classified as such.

Communication with the Holstein-Friesian Association of America revealed that no large tumorous growths have been called to their attention in





MULTIPLE LIPOMATOSIS IN TWO HOLSTEIN-FRIESIAN HERDS

Figure 17

The principal common ancestor in the pedigree was the paternal grandsire. There was no common ancestor in the maternal lineage within seven generations. The animal designated as "a" died in second lactation from injury to rear leg. At the time of death no lipomata were observed.

Holstein-Friesian or other breeds of dairy cattle. However, Thoele observed small lipoma capsulare in the escutcheon region of Holstein-Friesians[14]. These fatty tumors were approximately one inch horizontally and vertically and showed no tendency to increase with growth or aging of the animal, nor were they genetically implicated.

In the cases of multiple lipomatosis reported in this study, the economic value of the animals decreased considerably as the size and number of lipomata increased. In the two most aggravated cases in which fat deposition invaded the udder, serious malfunctioning of the mammary system resulted in lowered milk and butterfat production, and hand-milking was required. From a physiological standpoint, lipomatosis invades and displaces functional tissues throughout the body.

Nature of Inheritance

A pedigree chart of animals exhibiting lipomata is shown in Figure 17.

In herd A, the dam of the two animals exhibiting multiple lipomatosis (Figure 14) had four apparently normal offspring that reached maturity. Two of these normal offspring were full sibs to the animals shown in Figure 14; the other normal offspring were sired by two different bulls. The four normal animals were free of multiple lipomatosis and possessed high

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PROGRESSIVE MULTIPLE LIPOMATOSIS

Figure 16

The cow with this developmental trait is pictured at (*A*) three, (*B*) five, and (*C*) seven years. Although the animal ranked in the upper one percent of the breed in terms of butterfat production, her economic value decreased considerably as the size and number of lipomata increased.

productive traits. When this dam was mated to a bull from herd B tracing back to the common paternal ancestor in the two herds, she produced two normal offspring and two animals exhibiting lipomatosis.

In order to check the ancestral linkage in the two herds, pedigree information was compiled from the Holstein-Friesian Herd Books. The principal common ancestor in the pedigree was the paternal grandsire. No common ancestor in the maternal lineage within seven generations appeared in either herd. Thus, the afflicted animals in both herds have a common ancestor in the paternal lineage but do not possess a common maternal ancestry.

Further examination of the pedigree data revealed that common genealogy was prevalent on a within-herd basis. The dam of the two animals exhibiting multiple lipomatosis had two common ancestors in the third generation.

In herd B, two bulls were used to sire the 75 dams of the 75 daughters shown in Figure 17. In the third generation, these two bulls had the same paternal great-grandsire but they were unrelated through their maternal lines. They had an inbreeding coefficient of three and one-eighth percent as computed by the formula of Wright[15].

There is a possibility that a common recessive gene for this condition may have been passed to the afflicted animals in both herds. This supposition is doubtful because no common ancestors existed on a between-herds basis for seven generations in the maternal lines. A check beyond the seventh generation for common ancestry seems unwarranted as the mating of animals in the early stages of the breed development (before 1900) was on a close mating system due primarily to the small number of animals available and a lack of transportation.

The possibility remains that a recessive gene condition may exist within the herds. Herds A and B, on an individual basis, are closely related. The mating of animals within these two herds to the common paternal ancestor could have resulted in the condition of lipomatosis.

The mode of inheritance of multiple lipomatosis in humans is generally felt to be a simple Mendelian dominant[5, 12, 13]. The data presented herein follow a similar pattern. The condition of multiple lipomatosis is not an "all or none" situation and wide variations exist between affected individuals. Multiple lipomatosis could be a trait that is determined by many genes. Due to the range in lipomatosis occurrence, severity, and position on the animals' bodies in these two herds, there is a possibility that the occurrence of this malady is inherited as a Mendelian dominant with incomplete penetrance. This view could be tested with subsequent offspring from animals possessing multiple lipomatosis.

Summary

Multiple lipomatosis was observed in 22 animals in two herds of Holstein-Friesian dairy cattle. Lipomata were first observed at the age of three and one-half years and progressed extensively as the animals became older. In two high-producing cows, the fatty-like tumors caused serious malfunctioning of the mammary system resulting in lowered production yields. In no instance within the kindred did any of the lipomas become malignant.

It is suggested that multiple lipomatosis in cattle is inherited as a Mendelian dominant with incomplete penetrance, similar to the condition found in humans.

Literature Cited

1. Blom, E. and N. O. Christensen. Congenital absence of the epididymis ductus deferens or glandular vesicularis in the bull. *Bul. Department of Special Pathology and Therapy, Royal Veterinary and Agricultural College, Copenhagen, Denmark.* pp. 1-64. 1951.

2. Eaton, O. N. A summary of lethal characters in animals and man. *Jour. Hered.* 28: 320-326. 1937.

3. Eriksson, K. Hereditary forms of sterility in cattle. Biological and genetic investigations. *Lund Hakan Ohissons Boktryckeri.* 1943.

4. Hartung, E. W. The inheritance of a tumor in *Drosophila melanogaster. Jour. Hered.* 41:269-272. 1950.

5. Hellier, F. F. Hereditary multiple lipomata. *Lancet* 228:204-205. 1935.

6. Herman, H. A. and F. W. Madden. The Artificial Insemination of Dairy Cattle. A Handbook and Laboratory Manual. Lucas Brothers Publishers. Columbia, Missouri. 1953.

7. Johansson, I. Personal communication. 1959.

8. Kurzweg, F. T. and R. Spencer. Familial multiple lipomatosis. *Am. Jour. Surg.* 82: 762. 1951.

9. Mead, S. W., P. W. Gregory and W. M. Regan. An hereditary digital anomaly of cattle. *Jour. Hered.* 40:151-155. 1949.

10. Moulton, R. F. A symposium on mammary tumors in mice by members of the staff of the National Cancer Institute. *Am. Assoc. for Adv. of Sci.* No. 22. 1945.

11. Regan, W. M., P. W. Gregory and S. W. Mead. Hereditary strabismus in Jersey cattle. *Jour. Hered.* 35:233-234. 1944.

12. Shanks, J., A. W. Paranchych and J. Luba. Multiple lipomatosis. *Canadian Med. Assoc. Jour.* 77:881-884. 1957.

13. Stephens, F. E. and A. Isaacson. Hereditary multiple lipomatosis. *Jour. Hered.* 50:51-53. 1959.

14. Thoele, H. W. Personal communication. 1959.

15. Wright, S. Coefficients of inbreeding and relationship. *Amer. Nat.* 56:330-338. 1922.

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CONTENTS

T. E. Smith. The Cocos-Keeling Islands: A Demographic Laboratory.

Flann Campbell. Birth Control and the Christian Churches.

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